THE JOURNAL OF PERIODONTOLOGY

Vol. 10

January - July

1939

DENTAL LIBRARY

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY

VOLUME 10.
NUMBER 1.

120

RK 1
J855
v. 10

CONTENTS

The Historical Background of Periodontology
Biographical Sketches . . . . . ARTHUR H. MERRITT . 7

Progress in the Recognition of Etiologic Factors of Periodontal Diseases . A. W. BRYAN . . . 25

The Past and Future of Periodontia . PAUL R. STILLMAN . 31

The Responsibility Is Ours 37

Traumatic Occulsion and Gum Inflammation
BALINT ORBAN . . . 39

Twenty-Sixth Annual Meeting A. A. P. 47

An Experimental Study of the Use of Sodium Sulphid Solution in Treatment of Periodontal Pockets . . . . . FRANK E. BEUBE . . 49

Abbreviations for Dental Periodicals Published in the United States and Canada 56

Book Reviews 59

Publication in the JOURNAL OF PERIODONTOLOGY is not to be understood as endorsement by the Journal or the AMERICAN ACADEMY OF PERIODONTOLOGY of opinions expressed in any article, abstract or extract. The object of publishing the various views is to present them without prejudice to the reader for his own evaluation.

THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain therefore those papers of scientific and clinical interest which are presented at the annual meetings of that society. The proceedings of the 1938 annual meeting are published in part in this number; the remainder will appear in the July 1939 issue.

722804

DENTAL LIBRARY

If men could learn from history,
what lessons it might teach us!—
But passion and party blind our eyes,
and the light which experience gives
is a lantern on the stern which shines
only on the waves behind us.

—*Coleridge.*

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED THIRTY-NINE

[A SYMPOSIUM]

The Historical Background of Periodontology*

History is neither more nor less than biography on a large scale.—LAMARTINE.

BIOGRAPHICAL SKETCHES

By ARTHUR H. MERRITT, D. D. S., M. S., F. A. C. D., F. A. A. P.
New York City

NO ATTEMPT to record the achievements of those in the past who have contributed in one way or another to the evolution of periodontology can do justice to the subject. Nor can it be complete. It covers the whole range of recorded history, for in all ages of which there is any record, diseases affecting the supporting tissues of the teeth have been noted and much has been written about them and their treatment. With no knowledge of their etiology or differential diagnosis, attempts at treatment were, as might be expected, as ineffective as they were unscientific. It was not until within very recent years that any substantial progress can be said to have been made in the field which has come to be known as periodontology. Indeed, it was not until after the formation of the American Academy of Periodontology, that any organized effort was made to bring order out of the prevailing chaos. It is altogether fitting, therefore, as the Academy celebrates the Twenty-fifth Anniversary of its existence that it should pause in its deliberations to review the past with a view to a better understanding of the steps that have led up to the achievements of the present and at the same time to pay tribute to those whose contributions have made these achievements possible.

As one reviews that past it will at once become evident that little if any progress was made in the field of periodontology before the 18th century. From earliest times those charged with the treatment of disease had noted the presence in the mouth of a condition that caused teeth to loosen and fall out. Unsuccessful attempts at treatment were made, but nothing worthwhile seems to have been added to our knowledge of the subject before the days of Fauchard whose contributions to the field of periodontology cannot be said to have been conspicuous.

* Prepared by invitation and read at the 25th Anniversary Meeting of the American Academy of Periodontology at St. Louis, Missouri, October 20, 1938.

DENTAL LIBRARY

Pierre Fauchard 1678-1761

Pierre Fauchard, often referred to as the father of modern dentistry, was born in Brittany in 1678 and died in Paris March 21, 1761 at the age of 83.[1] In 1728 he published his celebrated work *Le Chirurgien Dentiste* in two volumes of 863 pages and with 20 full page illustrations. It went through two French editions and in 1733 was translated into German. This was probably the most comprehensive work on dentistry that had been published up to that time since it covered every phase of the subject as it was then known including orthodontia and oral hygiene. For over half a century it remained the standard work in dentistry.

It was not, however, until the publication of the second edition of his book in 1746 that Fauchard refers to a disease of the gums which he calls "a kind of scurvy." Describing it he says: "This disease, having no relation to the other parts of the body, attacks the gums, the alveoli and the teeth. Not only are the gums affected by it which are livid, swollen and inflamed, but those which do not show these symptoms as yet are not immune from this affliction. It is recognized by a yellowish almost white pus and by a little glutinous material, which is emitted from the gums when a rather heavy pressure is applied by the finger . . ." Regarding its treatment he says, "that those who have been treated for this disease by internal remedies, whether they are anti-scorbutic or not, have not been cured."

Pierre Fauchard

In the care of the mouth, he expresses disapproval of tooth brushes, recommending instead the use of small sponges or the end of a root of marshmallow, with which to cleanse the teeth and "fortify the gums." Dentifrices and mouth washes of a most complicated nature are also recommended. It is surprising to find in a book in so many respects in advance of its time the recommendation to "rinse the mouth every morning and also in the evening before going to bed with a few spoonfuls of one's own urine immediately after it has been emitted, always provided the individual be not ill."

Six chapters of Volume One, second edition, are devoted to the consideration of the anatomy, physiology and pathology of the gums and their treatment which includes the splinting of loose teeth. Fauchard's chief contribution to periodontia would seem to be his recognition of the part played by oral sepsis in causing diseases of the teeth and gums for he says: "Little or no care as to the cleanliness of the teeth is ordinarily the cause of all the maladies that destroy them." This was an important observation 200 years ago.

John Hunter 1728-1793

John Hunter, the celebrated physiologist and surgeon, was born in Glasgow, Scotland, February 13, 1728, the youngest of ten children. Though not a dentist he published two books on the subject of dentistry: *Natural History of the Human Teeth* (1771) and *Practical Treatise on the Diseases of the Teeth* (1778). He treats at length of the diseases of the alveolar process referring to two types of disease which he classifies according to whether there is a discharge of pus from around the affected teeth. The alveolar process is, in his opinion,

the seat of the disease. No remedy known to him was effective in its treatment. He died in 1793 at the age of 65.[2]

Bernard Bourdet

Bernard Bourdet, dentist to the King, is said to have published in 1757 the best book on dentistry since Fauchard. His gifts of literary and scientific culture, coupled with wide experience and an insatiable thirst for knowledge made him one of the outstanding practitioners of his generation. He seems to have been more interested in orthodontics than in periodontia though he refers to it and advocates the use of the actual cautery in the treatment of periodontal pockets as follows: "A cautery flat and light is needed, which is reddened in the fire and passed to the bottom of the space found between the gum and the root of the tooth. This is repeated two or three times, being careful to burn the gum on all of the faces which correspond to the root."[3] (Translated from the French.) He also wrote an excellent book on dental hygiene which was subsequently translated into German and Italian.[4]

Jean Jacques Joseph Serre 1759-1830

Of the dentists of the 18th century and the beginning of the 19th special mention should be made of Jean Jacques Joseph Serre.[5] Though born in Belgium his professional activities were carried on chiefly in Vienna and Berlin. In 1791 he published a book in Leipzig dealing extensively with diseases of the gums. Another book on oral hygiene dealt with the ways and means for maintaining the gums in health. This latter book met with much favor and went through two editions—1809 and 1812. Here again we find an author stressing the importance of mouth hygiene.

Kunstmann

In 1772 a man by the name of Kunstmann published a small booklet setting forth the treatment of pyorrhea by surgical measures. The nature of the operation as recommended by him is not quite clear, but seems to have consisted in surgically opening up the area to be operated upon or a removal of the involved gum tissue—probably a combination of both.* This seems to have been one of the first, if not the first, attempt to treat this disease by surgical interference of which there is any record. Serre refers to the operation as seen by him in the mouth of a patient operated upon by Kunstmann saying that "the roots of the teeth became exposed and the teeth appeared much longer which cannot be avoided with this operation as the gum tissue during the process of healing will recede to the area where the peridental membrane has not yet been destroyed. . . . This operation is very well devised, but tedious, as it has to be repeated at different times. If, instead, the entire pocket is cut away at once and the root cleansed, the incision will close readily. The ultimate good results of the operation are quite satisfactory: Kunstmann, however, has forgotten to speak of the retraction of the gingiva from the destroyed section of the pericementum: thus, one might be led to assume that a new membrane had formed which is not possible."[6]

* Have been unable to obtain further data.

S. Robicsek 1845-1928

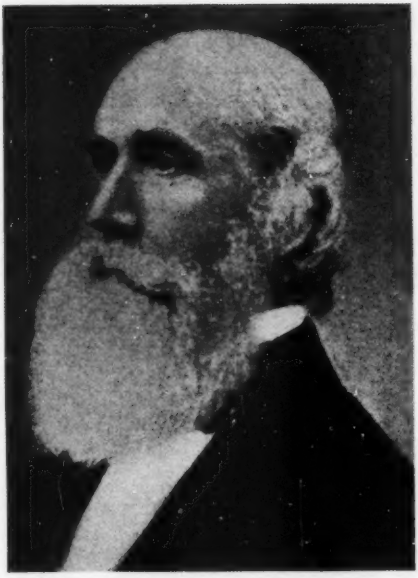
More than a hundred years later, another surgical procedure now generally known as a flap operation, was devised by a man named Robicsek and demonstrated by him before the Austrian Dental Society at Vienna in 1894. This operation consisted of laying back a flap of gum tissue over the area to be operated upon, curettement of the exposed root surfaces after which the gum flap was brought back into place and sutured. This operation was more or less limited to the upper anterior teeth. It was this operation which was subsequently introduced into this country by Zentler, who was a Rumanian by birth and may have been familiar with the operation as practiced in Europe.

Robicsek was born in Bajsa, Hungary, October 8, 1845, and spent his early life in Prague. In 1868 he was graduated in medicine from the University of Vienna. He later took up the practice of dentistry which he continued for the balance of his professional life. He occupied a distinguished place in the dental profession of Europe, numbering among his friends such men as Arkovy, Miller, Jenkins and Younger. His so-called flap operation seems to have been his chief contribution to periodontia. He died February 17, 1928 at the age of 83.[7]

John M. Riggs 1810-1885

It is to American dentistry that one must turn in his search for those who laid the foundation of modern periodontology. And first among these from a chronological standpoint as well as from the importance of his contribution to the subject, is the name of John M. Riggs of Hartford, Conn.

Riggs was born in Seymour, Conn., October 25, 1810, or 1811, and died in Hartford, November 11, 1885.[5] In 1837 he was graduated from Trinity College, Hartford, with a Bachelor's Degree. For two years he taught school and then began the study of medicine in the Jefferson Medical College, Philadelphia, but before graduation turned his attention to dentistry. His studies in this field were carried on under the preceptorship of Dr. Horace Wells of Hartford, Conn., in whose office Dr. Wm. G. T. Morton also studied dentistry. Very early in his career he became interested in the study and treatment of what was then called "scurvy of the gums,"—an interest which continued throughout his professional life. On several occasions he gave clinics demonstrating his method of treatment and was credited by his contemporaries with "originating and first publicly describing a new treatment for the cure of inflammation of the gums and absorption of the alveolar process, or the so-called 'scurvy of the gums' thereby saving and restoring to comparative firmness the loosened teeth." It is important in evaluating his contribution to periodontology, that we obtain a clear understanding of what his treatment consisted for on this point there has been and still is considerable confusion. This is due largely to his use of the term "surgical" as applied to his methods of treatment. Up to the time of Riggs practically all treatment was of a palliative nature. Application to the gums of some drug such as the tincture of myrrh, the use of astringent mouth washes, etc., was the usual treatment. When the teeth became so loose as to be troublesome they were extracted. What set Riggs apart from his contemporaries was his insistence that "scurvy of the gums" as it was then called could be cured and that its treatment was not therapeutic, but surgical. By this latter term he meant, treatment by the use of instruments. He did not surgically remove the gum tissues or make use of any of the present day operations employed in periodontia. His treatment was



John M. Riggs

nothing more or less than that of subgingival curettage, crude of course as compared with the same operation today, but the same in principle except that he emphasized curettement of the alveolar process. This operation he performed with instruments (6 in number) of his own devising. It is this use of instruments and his reference to their application as surgery, that explains the confusion which at present exists regarding his method of treatment.

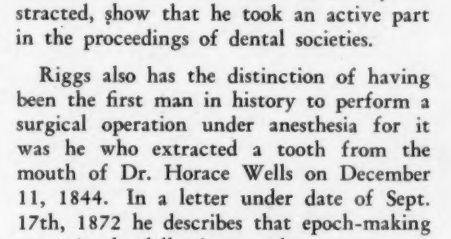
Curiously enough, G. V. Black *(Special Dental, Pathology* p. 66) falls into the same error for he says in describing Riggs' operation: "The gingivae and gum tissue were cut away sufficiently to remove all diseased tissue to the line of the attachment of the peridental membrane." This is incorrect. The operation as performed by Riggs is described in detail by G. A. Mills (*D. Cosmos* Feb. 1878 p. 92) which shows it consisted of a curettement of the involved root surfaces and border of alveolar process. It is referred to by Mills as a "process of scraping by which the roots of loosened teeth are freed from extraneous particles." (*D. Cosmos* Vol. 20:349-406. 1878.) The most prolific cause of failure was in his opinion lack of thoroughness which is probably as true of the present as it was in the

days of Riggs. That there were those in his day who made some use of surgery in the practice of periodontia is probable, for in one of his few published discussions he refers to those who remove "strips of the gum, from wisdom tooth to wisdom tooth" and characterizes such treatment as "simply barbarous."[9] He recognized but one periodontal disease which he divided into four stages as follows: "*First.* Where the margins of the gums show decided inflammatory action, with some absorption of its substance, and bleeding at the slightest touch of the brush. *Second stage.* Where the inflammation extends down over the thinner alveolar border, causing absorption of the bone, as well as the gum tissue, forming small pockets beneath the gum filled with pus. *Third.* Where the diseased action takes deeper hold, involving the thicker portions of the alveolar process, absorbing it most rapidly nearest the tooth, causing the tooth to sway back and forth for lack of most of its bony support. *Fourth and last stage.* Where the disease has swept away all the alveoli and much of the gum, the tooth being held in place by the conversion of the peridental membrane at the apex of the root into a tough ligamentous attachment."[10] Regarding its etiology and treatment he says: " . . . the teeth themselves, with their accumulated accretions and roughened surfaces, from whatever source derived, are the exciting cause of the disease, under consideration. The teeth in perfect polish and cleanliness, at and under the margin of the gums whether of animals or man, produce no inflamed action in that tissue. It can be artificially produced, however, by inserting a foreign body into or beneath its substance. If then, diseased action can be set up by a foreign body, artificially introduced, it can be arrested and cured by withdrawing the same. And, therefore, if the tooth becomes an extraneous body by reason of the accretions and concretions upon it, near and under the free margin of the gum, and inflammation ensues, as it certainly will, the true prophylactic treatment surely would be to thoroughly and carefully remove said concretion, tartar and roughness—polish the tooth and let Nature take care of the rest."[10]

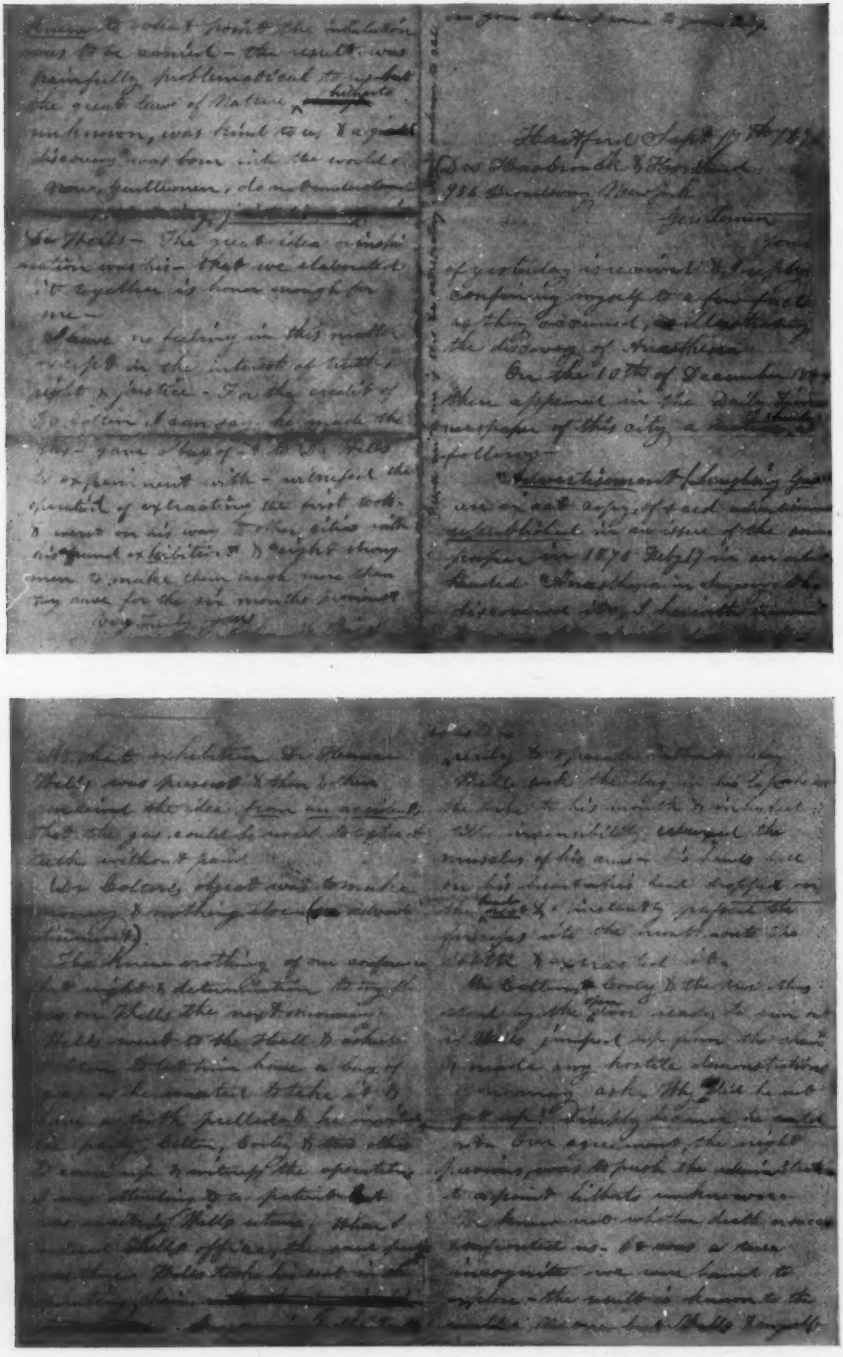
Because of the revolutionary nature of his teachings and his insistence that the disease was one which could be cured, he made an indelible impression on his generation and one which has continued down to the present. His name became known wherever dentistry was practiced, and was given by his contemporaries to the disease which he had done so much to emphasize.

Though Riggs was a frequent attendant at dental meetings he seems to have written but few articles on his favorite subject. One of these published in the *Penn. Journal of Dental Science* under the title of *Suppurative Inflammation of the Gums and Alveolar Process* was reprinted in *Johnston's Dental Miscellany* for Aug. 1878 with a foot note by the Editor which states: "This subject is exciting so much attention, both here and in Europe, that we reprint what a valued correspondent says is the only authoritative statement of the matter, from Dr. J. M. Riggs' own pen." This was read before the American Academy of Dental Science at New York on Oct. 20, 1875 and bears the name of the author "John W. (M) Riggs F. A. A. D. S., Hartford Ct." It is the only article by him I have ever been able to find. A few discussions, usually abstracted, show that he took an active part in the proceedings of dental societies.

Riggs also has the distinction of having been the first man in history to perform a surgical operation under anesthesia for it was he who extracted a tooth from the mouth of Dr. Horace Wells on December 11, 1844. In a letter under date of Sept. 17th, 1872 he describes that epoch-making event in the following words:



Envelope of letter reproduced on page 12



Reproduction of letter from John M. Riggs to Drs. Hasbrouck and Howland. See p. 13.

Drs. Hasbrouck & Howland,
956 Broadway, New York

Gentlemen:

Yours of yesterday is received and I reply, confining myself to a few facts as they occurred, illustrating the discovery of Anaesthesia.

On the 10th of December, 1844, there appeared in the Daily Times, newspaper of this City, a notice headed as follows:

"Advertisement, Laughing Gas," an exact copy of said advertisement republished in an issue of the same paper in 1870, Feb'y 17 in an article headed, "Anaesthesia in Surgery. Who discovered it?" I herewith transmit to you. It speaks for itself.

At that exhibition, Dr. Horace Wells was present and then and there conceived the idea *from an accident*, that the gas could be used to extract teeth without pain.

Dr. Colton's object was to make money and nothing else—(see advertisement).

He knew nothing of our conference that night and determination to try the gas on Wells the next morning. Wells went to the Hall and asked Colton to let him have a bag of gas, as he wanted to take it and have a tooth pulled, and he invited the party, Colton, Cooley and two others to come up and witness the operation. I was attending to a patient, but was awaiting Wells' return. When I entered Wells' office, the said parties were there. Wells took his seat in the operating chair. I examined the tooth so as to be ready to operate without delay. Wells took the bag in his lap—held the tube to his mouth and inhaled till insensibility relaxed the muscles of his arms—his hands fell on his breast—his head dropped on the head rest and I instantly passed the forceps into the mouth, onto the tooth and extracted it.

Mr. Colton, Cooley and the two others stood by the open door, ready to run out if Wells jumped up from the chair and made any hostile demonstrations. You may ask, "Why did he not get up?" Simply because he could not. Our agreement the night previous was to push the administration to a point hitherto unknown.

We knew not whether death or success confronted us. It was a *terra incognita* we were bound to explore—the result is known to the world. No one but Wells and myself *knew* to what point the inhalation was to be carried—the result was painfully problematical to us but the great law of Nature, hitherto unknown, was kind to us and a grand discovery was born into the world.

Now, Gentlemen, do not understand me as claiming *joint-discovery* with Dr. Wells. The great idea or inspiration was his—that we elaborated it together is honor enough for me.

I have no feeling in this matter except in the interest of truth, right and justice. For the credit of Dr. Colton I can say, he made the gas, gave a bag of it to Dr. Wells to experiment with—witnessed the operation of extracting the first tooth and went on his way to other cities with his "grand exhibition" and "eight strong men to make them laugh more than they have for the six months previous."

Very truly yours,

(Signed) JOHN M. RIGGS[11]

In recognition of his contribution to dentistry the degree of Doctor of Dental Surgery was conferred upon him by the Baltimore College of Dental Surgery in 1879.[12] For several years he was clinical instructor in the Harvard Dental School. In 1881 he attended a meeting of the International Medical Congress in London where he gave lectures and clinical demonstrations on his method of treating "scurvy of the gums."[13]

To Riggs more than to any other man of his generation periodontology is under obligation, for he was the first in this country to insist that *"Suppurative Inflammation of the Gums"* as he called it could be cured and that its treatment was surgical (that is by the use of instruments) and not therapeutic. In this respect he was ahead of his generation. He seems to have been a man of strong individuality, an independent thinker and fearless in the expression of his convictions. He died at Hartford, Conn. November 11, 1885 in his 76th year. The following letter written to one of his patients and now in my possession, would seem to indicate that he was not lacking in self-assurance or in appreciation of the importance of his contribution to the progress of dentistry:

Hartford, April 23, 1885

Miss C. A., Somaine

Dear Miss:

Your information relative to your teeth was quite correct—A very severe cold upon my lungs has delayed my answer till today—this being my second letter written for two months—At the time you was in Hartford, I was in business in Church Street. I had not retired from practice and never have except compelled temporarily from sickness. The treatment originated with me and owing to its difficulty of application, meets with great opposition. Gen. Grant's trouble probably originated in this disease and has been aggravated by the nicotine of his cigars and may preclude recovery. Yet knowing what I do of cases similar in my practice the chances are that he will recover. Physicians know very little of the disease and the dentists less. They are not to be censured for it is fifty years ahead of the age.

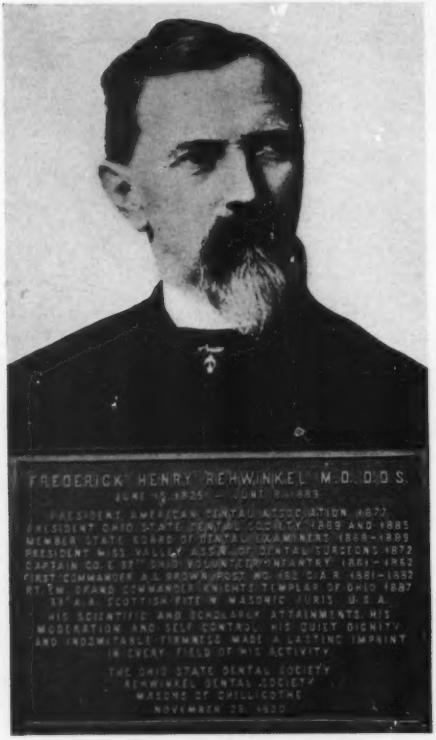
In a week, or thereabouts, I shall have gained such strength as to meet many appointments for treatment from other cities. I can not write any more at present, only to say I can tell you when I see you the true condition of your mouth.

Dr. Taylor recovered from his sickness and returned here to practice, and to be frank with you I could not recommend his treatment of your case, yet you must do as you please and—In a week or 10 days I shall be strong as I am gaining rapidly.

Hoping you will meet with entire success in retaining your teeth I remain

Very truly yours

JOHN M. RIGGS



F. H. Rehwinkel, M. D., D. D. S., 1825-1889

Dr. Rehwinkel whose chief contribution to periodontology seems to have been the introduction into this country of the term "pyorrhea alveolaris" in 1877, was born in Celle, Hanover, Germany, June 15, 1825. After graduating in Homeopathic medicine he emigrated to the United States in 1848 or '49. For several years he continued the practice of medicine, first in Natchez, Miss. and afterwards in Cincinnati and Portsmouth, Ohio, but becoming interested in dentistry he took up its study and was graduated from the Baltimore College of Dental Surgery in 1854. He located in Chillicothe, Ohio, where he carried on the practice of dentistry up to the time of his death in 1889.[15] The term "pyorrhea alveolaris" was not original with Rehwinkel having previously been used by European writers a fact doubtless known to him. It is reported that the term was first given to this disease by one named Toirac (1823).[16] His paper on pyorrhea alveolaris (1877) was said to have been the best that had appeared on the subject up to that time though it abounded in inquiry rather than the announcement of any new principal of pathology or treatment.[17] He died June 8, 1889.

G. V. Black, M. D., D. D. S., Sc. D., L. L. D., 1836-1915

Though Dr. G. V. Black's formal education was limited to attendance at a country school for a few months during the winter time, he had the distinction of possessing four doctors degrees, all honorary, including his medical and dental degrees. He was a life long student and a prolific writer on many phases of dentistry. It was as a research worker, teacher and writer that he made his chief contributions to periodontology. As a clinician in this field his experience seems to have been somewhat limited. He was more or less skeptical regarding the treatment of what he at one time called *"phagedenic pericementitis"* and later changed to *"chronic suppurative pericementitis."* After enumerating the adverse conditions present in these cases he says: "how shall we expect pockets to heal, reattachment of the soft tissues to the cementum to occur, and permanent cures of this condition to be made? As a matter of fact, such cures do not occur, notwithstanding the reports of successes."[18] It was his opinion that the disease was chiefly local, that gingivitis always preceded the formation of pus pockets and that serumal calculus was an important causative factor.[19] He summed up his conclusions in the statement that suppurative detachments of the pericementum were permanent detachments.

William J. Younger, M. D., D. D. S., 1838-1920

Dr. William J. Younger was born of English parents in Santiago, Chile, October 9, 1838, removing to San Francisco, California, with his parents when he was ten years of age.[20] In 1862 he was graduated from the Medical Department of the University of the Pacific and immediately began the practice of medicine which he continued for about a year. He then took up the specialty of oral surgery in which he

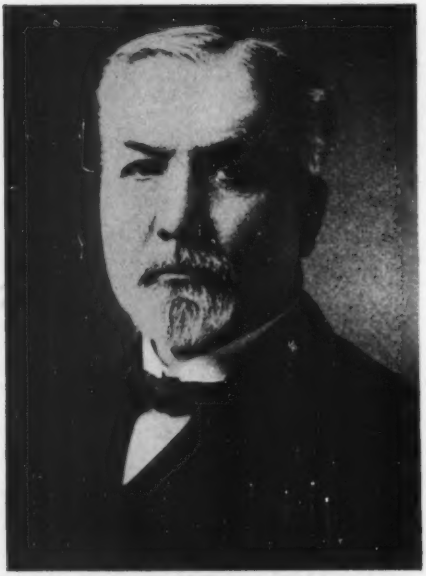
William J. Younger

became well known for his treatment of what was then called pyorrhea alveolaris and for the implantation of teeth in artificially formed sockets in the jaws. This latter operation he demonstrated at the Ninth International Medical Congress at Washington, D. C. in 1887 where it was accredited with being "the original operation of the Congress of that year." In 1897 he removed to Chicago and in 1900 took up his residence in Paris where he continued in practice up to the time of his death July 22, 1920. It was his belief that pyorrhea was of purely local origin.[21] He was insistent upon thorough curettage of involved root surfaces and the subsequent application of lactic acid to the pockets. By the application of these measures he claimed he was able to cure 96% of his cases.[22] That which distinguishes him in the field of periodontology is that he was among the first, if not the first, to claim that reattachment took place in cases properly treated. On this point he said in 1892: "The treatment of pyorrhea alveolaris by the method recommended by myself several years ago is meeting with unabated success in my hands. Teeth hanging by apical attachment only, have been rendered firm, and tissues of the diminished sockets made so to contract and unite with the roots, that an instrument could not be introduced between them without force, and causing pain and bleeding. I am satisfied that attachment takes place in these cases, otherwise the teeth could not become so firm and the gums and tissues of the socket cling so to the tissues of the root. It is, therefore, an easy matter for direct union to be established between these healthy, living tissues, when the partition of calculus is thoroughly removed from the root, and a granulating surface produced in the environing soft tissues of the gingivus."[22] Like Riggs he depended for success upon the skillful use of instruments though his technique was more refined and he placed less emphasis upon the curettage of the border of the alveolar process. He devised the instruments he used, many of which have never been improved upon. He was emphatic in claiming that the *sine qua non* in treatment was *complete* removal of all calculary deposits upon the surface of the exposed cementum. His chief claim to distinction in the field of periodontology is in his insistence that reattachment could be brought about in advanced cases of pyorrhea when properly treated.

David D. Smith, M. D., D. D. S., 1839-1920

Another man who made an indelible impression upon his generation, comparable in many respects to that made by Riggs and Younger was David D. Smith.[23] He was born in Greene County, N. Y., July 1, 1839. At nine years of age he removed with his parents to Pennacook, N. H. where he received his early education in the public schools and in the Boscawen Academy. In 1859, following a brief period of teaching, he took up the study of dentistry in the office of a dentist in Cambridge, Mass. He subsequently began practice in Pennacook and is said to have made the first set of teeth on a vulcanite base worn in the State of New Hampshire. He later entered the Philadelphia Dental College from which institution he was graduated in 1867. In the same year he was made Professor of Mechanical Dentistry in his Alma Mater. While engaged in teaching, he attended the Jefferson Medical College in Philadelphia and was graduated M. D., in 1878.

In the early part of his professional life he seems to have been engaged in general



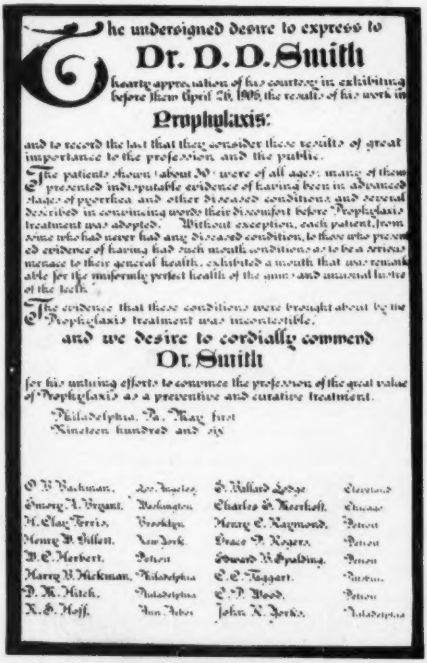
David D. Smith

practice. He later became interested in what he called "oral prophylaxis." This he defined as a "surgical treatment of the mouth and teeth, as distinguished from the administration of a medicament or a therapeutic remedy, for the prevention and arrest of inflammatory or diseased conditions of the oral cavity."[21] This consisted of the "careful and complete removal of all calcic deposits, inspissated secretions, bacterial placques, and all accretions which gather on the surfaces of the teeth, and between them, especially at the gum margins: followed by thorough polishing of all tooth surfaces by hand methods." By the application of these principles to daily practice he claimed it was possible to bring about almost complete immunity to decay; great improvement in color and appearance of the teeth; diminished sensitiveness of the dentin; tightening of teeth that had become loose; relief from sensitiveness of the gums; cleanliness and comfort of the mouth, and improvement in the character of the breath.

To accomplish this he insisted upon monthly prophylaxis. His efforts were mainly directed toward prevention though treatment of periodontal diseases, naturally formed a part of his practice. How successful he was in the treatment of pyorrhea in its more advanced stages there is little in the written record to indicate.* Regarding home care by the patient he says: "My patients are always instructed, in connection with the prophylaxis treatment, to use the brush with a vigorous horizontal movement, even when the gums and alveolar process have necrosed until the festoons have become everted, and to continue the brushing until the teeth are not only brushed but *cleansed*."[22] This was written in 1904. It has a strange sound in 1938.

His chief contribution to periodontology seems to have been his insistence upon the importance of oral prophylaxis. This he systematized and brought to a high art. Dr. Smith was possessed of all the qualities of a crusader. Thoroughly persuaded of the truth of his convictions, he upheld them on all occasions with the fervor of an evangel. He probably did more than any other man of his generation to popularize and make known to the profession and to the public, the exceedingly important part which oral cleanliness plays in the prevention of all dental diseases. He died June 3, 1920.

*



The undersigned desire to express to

Dr. D. D. Smith

a hearty appreciation of his courtesy in exhibiting before them April 20, 1906, the results of his work in

Prophylaxis:

and to record the fact that they consider these results of great importance to the profession and the public.

The patients shown (about 30) were of all ages; many of them presented indisputable evidence of having been in advanced stages of pyorrhea and other diseased conditions and several described in convincing words their discomfort before Prophylaxis treatment was adopted. Without exception, each patient, from nine who had never had any diseased condition, to those who presented evidence of having had such mouth conditions as to be a serious menace to their general health, exhibited a mouth that was remarkable for the uniformly perfect health of the gums and unusual luster of the teeth.

The evidence that these conditions were brought about by the Prophylaxis treatment was incontestible.

and we desire to cordially commend

Dr. Smith

for his untiring efforts to convince the profession of the great value of Prophylaxis as a preventive and curative treatment.

Philadelphia, Pa., May first
Nineteen hundred and six

G. V. Buckman,	Los Angeles	S. Ballard Dodge	Cleveland
Emery A. Bryant,	Washington	Charles S. Koerbel,	Chicago
H. Clay Ferris,	Brooklyn	Henry C. Raymond,	Detroit
Henry B. Allen,	New York	Bruce D. Rogers,	Detroit
W. C. Herbert,	Detroit	Edward B. Spalding,	Detroit
Harry B. Hickman,	Philadelphia	C. S. Taggart,	Detroit
D. M. Hitch,	Philadelphia	C. P. Wood,	Detroit
N. S. Hoff,	Ann Arbor	John N. Jerks,	Philadelphia

**"We were prepared to see good results, but were greatly surprised to see such a uniform lot of perfectly healthy and clean mouths, most of which had been redeemed from what had evidently been desperate cases of pyorrhea. We have sometimes been tempted to conclude that pyorrhea alveolaris was one of the diseases that would have to be classed as incurable. But since seeing these results, we have taken new courage and will henceforth not despair of even the desperate conditions which sometimes come to us."*—N. S. HOFF, Editorial. *D. Register*, 60 (6):271. June, 1906.

Levi C. Taylor 1841-1922[26]

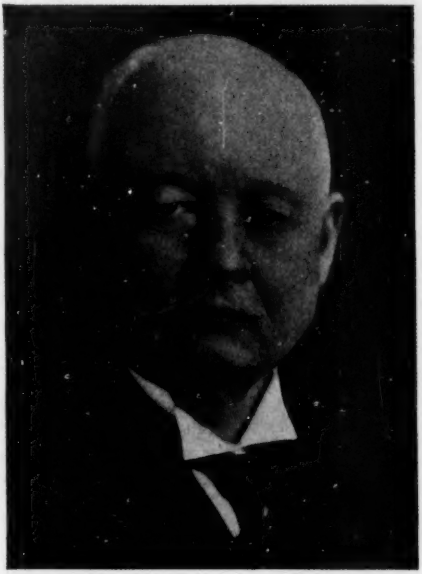
A contemporary of Riggs and for several years associated with him was Levi C. Taylor, born in Lempster, N. H. December 12, 1841. He studied dentistry in the office of Dr. George Bowers of Springfield, Vt., where he served an apprenticeship of several years. In 1868 he established himself in practice in Holyoke, Mass., removing to Hartford, Conn. in 1875, where he became associated with Dr. Riggs. For many years he was one of the leading exponents of preventive dentistry and like his protagonist, D. D. Smith, preached and practiced the gospel of oral prophylaxis. Though greatly handicapped by deafness in his later years, he was an enthusiastic and regular attendant at dental meetings and took advantage of every opportunity to proclaim the importance of oral hygiene in the prevention and treatment of periodontal diseases. He died at Hartford, Conn. February 8, 1922 in his 81st year.

Eugene S. Talbot, D. D. S., M. D., L. L. D., M. S., Sc. D., 1847-1924

Dr. Eugene S. Talbot was born March 8, 1847 at Sharon, Mass. the second of ten children.[27] He was educated in the public schools and at Stoughtonham Institute. In 1870 he began the study of dentistry in a private office. He subsequently entered the Pennsylvania College of Dental Surgery from which institution he was graduated in 1872 and immediately began practice in Chicago. In 1878 he took up the study of medicine at the Rush Medical College and was graduated M. D. in 1880. All his life he was a staunch believer in the medical education of the dentist and did what he could to bring it about.

In 1881, with the assistance of others he organized the Section of Stomatology in the American Medical Association and for thirty-three years was its secretary.

In addition to an active dental practice he carried on extensive researches along many lines. He is credited with having written 13 books and approximately 200 monographs.[28] One of these books *Interstitial Gingivitis or so-called Pyorrhea Alveolaris*, was published in 1899. In it he emphasized the transitory nature of the periodontal tissues and expresses the belief that this predisposed these tissues to disease.



Eugene S. Talbot

He also reviews the different theories that have been put forward regarding pyorrhea. The causes of *Interstitial Gingivitis* he divides into two groups—predisposing and exciting—the latter mostly local. In the brief chapter pertaining to treatment he is inclined to be pessimistic. "The disorder" he says, "responds quickly to treatment at its onset. Later, its complications and the extent of structure involved render treatment very inefficacious, and always insure the loss of the teeth." Treatment as recommended consisted in the removal of calculus, the application to the gums of tincture of aconite and iodine and their massage. In this latter operation he says, "The patient should be instructed with this single idea in view that the gum margin is to be exercised and stimulated and not the teeth which must be ignored."[29] Regarding mouth washes he says: "During the entire process of treatment (indeed before the treatment begins) a suitable mouth wash should be used two or three times a day. This should be composed of a germicide antiseptic, disinfectant, and deodorant, and should be used at time of massage." A study of Dr. Talbot's contribution to the science of periodontology forces one to the conclusion that

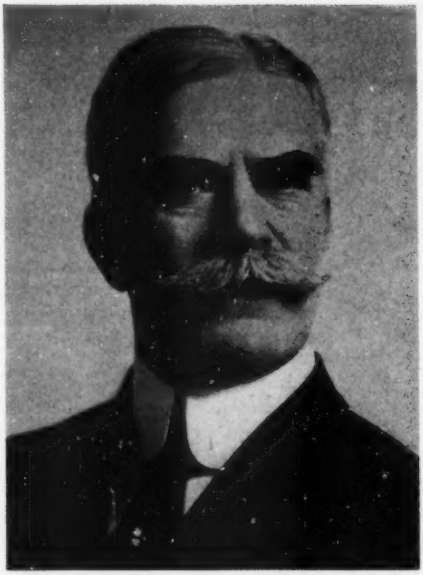
he was a theorist rather than a successful clinician—a seeker for the truth rather than a discoverer.

It was in 1903 that I first became interested in periodontia. In my search for information I purchased and read with much interest Talbot's book on *Interstitial Gingivitis*. Feeling a little disappointed but not being certain I was right in my estimate of its worth, I appealed to Dr. James Truman for his appraisal of the book and under date of March 8, 1904 he wrote me as follows: "I do not feel equal to doing justice to Talbot's *Interstitial Gingivitis*. I have, of course, read it and greatly appreciate the work done by himself and assistants, but I do not feel that much light has been thrown upon the etiology of the subject and the student and practitioner will leave the book, if like myself, entirely at sea in regard to treatment. Dr. Talbot is a warm personal friend of mine but I must say he does not seem to my mind to complete his subject. I do not either, like his name. Pyorrhea Alveolaris is not gingivitis whatever else it may be and calling it such seems to cast a cloud over his otherwise excellent histological work." He ends his letter by saying: "I cannot recommend you to any very satisfactory writings on this subject. They are all tinctured with the personal equation, my own included. To my mind it is a local disease, aggravated it may be, by systemic conditions, age, and bacterial invasion. But that I cannot enter upon here."

John Deans Patterson, D. D. S., F. A. C. D., F. A. A. P. 1848-1930

In 1885, the year in which Riggs died, Dr. John D. Patterson published a paper entitled "The Catarrhal Nature of Pyorrhea Alveolaris" in which he expressed the belief that pyorrhea was a form of catarrh.[30] Again in 1888 in a paper entitled "Points in the Etiology of Pyorrhea Alveolaris"[31] he re-affirms this belief in the light of further observations saying "I believe the disease is in every instance a true catarrh." As late as 1917 he seems to have held to this opinion though he modifies it somewhat by stating that "no doubt catarrh at times is the inception of pyorrhea."[32] He was an insistent advocate of thorough home care of the mouth.

Dr. Patterson was born in Clear Creek, Ashland County, Ohio, on February 9, 1848. He received his early education in the local schools and Savannah Academy.[33] For three years he studied dentistry in private offices and after a brief period of practice in Savannah, Ohio, he removed to Lawrence, Kansas, where he continued in practice until 1884, at which time he removed to Kansas City. He was one of the organizers in 1881 of the Kansas City Dental College, graduating from that institution in 1883. He was president of the Kansas and Missouri Dental Associations and also of the, American Dental Association, in 1893. He was editor of the *Western Dental Journal* for twenty years and was regarded as an important factor in the development of dentistry, especially in the middle west. He wrote several papers on the subject of pyorrhea and appears to have been an operator of more than usual skill. He was a member and fellow of the American Academy of Periodontology. He died at Kansas City, January 12, 1930.



John Deans Patterson

Robert B. Adair, 1849-1934

Among those from the South who contributed to the progress of periodontia was Dr. Robert B. Adair of Atlanta, Georgia. He was born in Jackson County, Georgia,

May 27, 1849, obtaining his early education in private schools and in Martin Institute. He began the study of dentistry in 1870, graduating from the Baltimore College of Dental Surgery one year later, and opened his office for the general practice of dentistry in Gainesville, Georgia, where he remained until 1895 when he removed to Atlanta. He was a great admirer of Riggs and throughout his professional life applied in his practice the principles of treatment as laid down by him. Dr. Adair was one of the pioneers in the field of periodontology and was a successful practitioner for more than sixty years. He died December 26, 1933., in the 86th year of his age.[34]

M. H. Fletcher, D. D. S., M. D., M. S. 1849-1914

In a paper entitled "Alveolitis—the Disease of Which Pyorrhea Alveolaris is one Stage," Dr. Fletcher states that in presenting the subject it is his desire "to view it from the standpoint of those who believe the affections of the alveolar process to be primarily a bone disease" because as he says, "its symptoms, progress, pathology and termination, as well as its systemic features, seem to be identical with diseases of bone in other parts of the body and not primarily a disease of the periosteum, peridental membrane, and gums as regarded by many. . . . In my opinion, the initial, exciting cause, in at least 9/10 of the cases, is the formation of calcareous deposits about the necks of the teeth."[35] Treatment recommended by him consisted "in the use of delicate bone curettes and suitably shaped burs for removing dead bone from about the neck and roots of affected teeth." He frequently refers to bone about teeth affected with periodontoclasia as "dead" or "necrotic." He was also insistent that "one of the most important parts of the treatment consists in teaching the patient how to keep the teeth free from calcareous deposits."

Dr. Fletcher was born in Richmond, Ind., September 18, 1849. He attended the district school until the age of fourteen at which time he was apprenticed to a jeweler for four years. He began the study of dentistry in the office of a preceptor in 1877. In 1879 he entered the Ohio Dental College from which he was graduated the following year. It is said, "He was graduated from the Miami Medical College in 1884 having done all dental and medical college work while practicing dentistry."[36]

He was for many years engaged in research and in 1895 after a course in the National School of Electricity was given a masters degree in science. Most of his professional life was spent in Cincinnati, Ohio. He died March 26, 1914 in the sixty-fifth year of his age.

Henry Barnes, M. D. 1851-1932[37]

Dr. Barnes was born in England, April 15, 1851, and came to this country with his parents two years later. In 1870 he moved to Cleveland, Ohio, where for two years he studied dentistry in the office of Dr. John Stephan. After a four years practice in Michigan, he returned to Cleveland and in 1879 was licensed to practice in the State of Ohio. In 1892 he was appointed professor of dental anatomy in the Homeopathic Hospital College where he received the honorary degree of M. D. For many years he was interested in oral prophylaxis saying that he knew of no single measure comparable to it in immediate beneficial results. He developed a toothbrush technique which he called "Contour cleansing." This consisted of what might be called interdental tooth brushing and gum stimulation. The effect he says is "immediate and astounding; gingivitis disappeared as if by magic, spongy gums were rapidly healed and pyorrhea responded to treatment as never before."[38] To accomplish this he used three brushes of different designs, all small, one having only a single row of bristles, which he says should be pliable, that is "softened stiff bristles *not soft* bristles." Dentifrices he regarded as unnecessary. Massage of the gums with the fingers was recommended in the post-operative treatment of pyorrhea. He died in 1932 in the 82nd year of his age.

Frank A. Pague, D. D. S. 1859-1930

Dr. Frank A. Pague at one time a member of the American Academy of Peridontology was born in Carlisle, Pa., May 20, 1859. Soon after graduation from the Pennsylvania College of Dental Surgery in 1889 he removed to San Francisco. Through his association with the late William J. Younger he became interested in periodontia and for many years was a recognized leader in this field on the Pacific

Coast. For seventeen years he was president of the California Academy of Periodontology. After the San Francisco earthquake and fire in 1906 in which he lost everything, he removed to Oakland. Seven years later he returned to San Francisco where he took up the practice of periodontia as a specialty. He was in his later years an enthusiastic exponent of the surgical treatment of periodontoclasia. He died March 5, 1930.[37]

Julian Smith, D. D. S. 1863-1931

Dr. Julian Smith was born in Charleston, S. C., April 5, 1863. In 1883 he was graduated from the Baltimore College of Dental Surgery and immediately engaged in practice in his native city. Twenty years later he removed to Austin, Texas, where he continued in general practice until 1916, at which time he removed to Dallas where he took up the practice of periodontia as a specialty. He was a clinician of more than average skill, using in his work instruments of his own design. He was a member of many dental organizations and in 1928-1929 was president of the American Academy of Periodontology. He died March 17, 1931.[39]

Jules J. Sarrazin, D. D. S., 1865-1931

On October 15, 1865, Dr. Jules J. Sarrazin was born in the City of New Orleans. He was educated in the Jesuits College, Tulane University, from which he was graduated in 1883. Four years later he graduated from the Baltimore College of Dental Surgery and began practice in the City of his birth which he continued up to the time of his death, September 14, 1931.[40] For many years he was engaged in the practice of periodontia. He was especially interested in oral prophylaxis and gave much thought to methods of tooth brushing, for which he devised brushes of special design.[41] In 1918-1919 he was president of the American Academy of Periodontology.

J. W. Jungman, D. D. S. 1867-1934

In February, 1914, a small group of men and women came together in the office of Dr. J. W. Jungman in Cleveland, Ohio, to consider the formation of an organization in periodontia.[42] Dr. Jungman was at that time engaged in the practice of periodontia and became one of the charter members of the American Academy of Periodontology which had its birth in his office. He continued an active member up to the time of his death in 1934. He was of a retiring nature and took relatively little part in the proceedings of the Academy. For many years he was engaged in the practice of periodontia and was reputed to be a clinician of more than average skill.

Alfred C. Fones, D. D. S. 1869-1938

Another whose contribution to periodontia was largely along the line of oral prophylaxis was Dr. Alfred C. Fones of Bridgeport, Conn., in which city he was born December 17, 1869. In 1890 he was graduated from the New York College of Dentistry. He originated the idea of instructing young women to give prophylactic care of the teeth and in 1913 established in his own office the first school for dental hygienists. He prepared with the coöperation of others, a text book for their instruction and all his life was interested in the promotion of this phase of dental practice. For this work he became famous throughout the world. Many honors were heaped upon him among them the award of the Jenkins, Jarvie and Fauchard medals. He died in his native city, March 17, 1938.[43]

Alfred C. Fones

Sidney J. Rauh, D. D. S. 1873-1935

Dr. Sidney J. Rauh was throughout his professional life interested in the social responsibilities of dentistry. He was an ardent believer in the importance of oral hygiene. No one of this generation did more than he to educate the dental profession and the public to the value of preventive dentistry as a health measure. He was concerned for the dental care of children and the underprivileged and was always ready and willing to give of himself and his talents in order that they might be better served. To him, perhaps more than to anyone else in this country, the oral hygiene movement owes its success. He was always in the forefront of any movement to educate the public to the importance of preventive dentistry. He did much to make Cincinnati the most dentally minded city in the United States. He was in very truth a preacher of the gospel of oral health and as such he will always be remembered. He was an enthusiastic member of the American Academy of Periodontology and took an active part in its proceedings. His death on November 18, 1935, robbed the Academy of one of its most unselfish and dependable members. Few, if any, can take his place in the work to which he gave so much of himself.[44]

Robin Adair, B. S., D. D. S., M. D. 1878-1929[45]

Among those who have made notable contributions to the science and art of periodontia was Robin Adair of Atlanta, Ga. He was born in Gainesville, Ga., February 27, 1878 the son of Dr. and Mrs. R. B. Adair. In 1899 he was graduated from the University of Georgia as Bachelor of Science and in 1902 in dentistry at the Southern Dental College of Atlanta. He then took up the study of medicine in the College of Physicians and Surgeons also at Atlanta, graduating in 1904. Soon after he began the practice of dentistry, he became interested in periodontology to which he devoted many years of his professional life, as practitioner, student and author. He possessed one of the best dental libraries in the South. In 1914 he published a book entitled *Practical Oral Hygiene, Prophylaxis, and Pyorrhea Alveolaris,* which he says was "written for those dentists who desire practical information on the subject." It contains 327 pages and includes much that is of practical value. Dr. Adair was a member of many professional organizations, (dental and medical) and took an active part in the organization of the Southern Academy of Periodontology. In 1928 he founded the Atlanta School of Oral Hygiene for the training of dental hygienists, which since his tragic death on April 26, 1929 has been named for him. He was at the time of his death Vice-President of the American Academy of Periodontology.[46]

Gillette Hayden, D. D. S., F. A. A. P. 1880-1929[47]

No study of the history of periodontology would be complete which did not include the name of Gillette Hayden. Few have made more notable contributions to its progress and no one was ever more interested in its success or gave to it a more unselfish service. Almost from the day of her graduation from the Dental Department of the Ohio State University in 1902 she seems to have been possessed of an intuitive sense of the importance of periodontia in the practice of dentistry. Never doubting that it represented the very cornerstone in oral health service, she decided very early in her professional career to devote her life

to its promotion. Accordingly she spent a year in post-graduate work at the Northwestern University Dental School in Chicago and later spent three years in Dresden in the active practice of what was then known as oral prophylaxis. She then opened an office in Columbus, Ohio, where she continued the same type of practice as a specialty.

Not satisfied with her personal achievements in this field, she set about the formation of an organization in which men and women, interested in this branch of dental practice might come together for mutual improvement and at the same time to undertake a campaign of education among general practitioners. With this in mind and with the coöperation of Dr. Grace Rogers Spalding who shared her vision and enthusiasm, an effort was made at the meeting of the National Dental Association at Kansas City in 1913 to enlist the interest of others in the proposed project. The only encouragement they received was "You women go ahead and organize and we will join." And this they proceeded to do.

In December of that year while in attendance at the meeting of the Ohio State Dental Society and later in Columbus and Detroit, these two women with little encouragement and less coöperation, completed their plans and sent out a call to a limited number of practitioners to meet in Cleveland in February 1914, at which time an Organization Committee was formed. Those responding were Doctors Clyde M. Gearhart, A. C. Hamm, Gillette Hayden, J. Herbert Hood, J. W. Jungman, John Oppie McCall, and Grace Rogers Spalding. After discussion it was decided to form a permanent association and to call a meeting in Cleveland for May 23 of the same year. In response to invitations, fourteen attended. An organization, to be known as The American Academy of Oral Prophylaxis and Periodontology was formed and Dr. Austin F. James was elected president. The name was subsequently changed to that of The American Academy of Periodontology. A year later Dr. Hayden was elected president. She also has the distinction of being the second person to receive the Academy fellowship. Her interest in the affairs of the Academy never faltered. She served on many committees and with one exception was never absent from its councils. The present status of periodontia is largely due to her vision and unceasing labors. No one can overestimate her part in its progress. She discharged well her obligations to her profession and to society. It is meet and right that the Academy should dedicate its Journal to her memory for it, too, represents one of her ideals. Dr. Hayden was born near Greenville, Florida, March 2, 1880 and died at Columbus, Ohio, March 27, 1929 in the 49th year of her age.[49]

* * *

In this incomplete review of the background of periodontology, there are recorded the names of twenty-five who in the past have made notable contributions to its progress. There are others, who though less noteworthy, have also played their part in its advancement. To name them all, would be difficult if not impossible. Among this latter group might be mentioned the names

Windsor County.

DENTAL NOTICE FOR 1832.

THE Subscriber flatters himself that the success which has crowned his

Operations on Teeth,

during a residenee of eight years in this village, is not unknown to a majority of the inbabitants of Windsor County. He thinks too, that he has not only kept up with the progress of improvement in his profession, but humbly believes that he has assisted materially in its advancement. During the past year he has constructed *Sixteen Whole Sets of Artificial Teeth*, besides numerous smaller pieces. Every meterial used is of the very purest quality ever employed by Dentists. Persons in need of any Dental Operation whatever, are assured that it will be done by the subscriber equal to any performance in the cities; and he requests those unacquainted with his work to address him by mail, when he will give them *references* as near their own residence as possible. Many of the whole sets made by him during the past year will resist a downward pull of 25 lbs without starting from the jaws. Even a *single tooth* is now put in on the atmospheric principle with success

A GOOD OFFER!

All persons residing at a greater distance than ten miles from this village, coming to the subscriber for any dental operations whatever, are informed that a discount of five per cent. will be made to them on the amount of their bill. All residing more than twenty miles from Woodstock will be allowed a discount of eight per cent. All more than thirty miles distant ten per cent.

This offer is made because the subscriber has determined to discontinue his professional visits to other towns. His great increase of business compels him to this course, and he hopes it will be as much for the benefit of his patients generally, as for himself. Rely therefore on finding him hereafter,

ALWAYS AT HOME.

Persons at a distance are invited to make such enquiries by mail, in regard to the various operations and their prices, as they choose, which will be answered with pleasure. HENRY S. CHASE, M. D.

Woodstock, Vt., Dec. 1, 1831. eop 6m

of Henry S. Chase[49] whose observations on tartar formation made more than 65 years ago will still bear study; A. W. Harlan, whose interesting chapter "Calcareous Deposits on the Teeth"[50] published in 1887 and illustrating instruments for their removal designed by seven different men is worthy of recognition as well as are his eighty other literary contributions relating to periodontia; George A. Mills[51] and C. G. Davis[52] both ardent disciples of Riggs; and M. L. Rhein, who attempted a classification of pyorrhea according to etiologic factors.[53] In more recent years are to be found the names of N. S. Hoff, F. H. Skinner, George H. Sprinkle, George H. Mengel and Richard Walden, all members of the Academy. And there are others. These too played their part. And in the great temple of human endeavor what matter who puts on the cope stone and the shining pinnacle. What matter who does the work and gets the credit so long as the work is done. And it is being done, thanks to the patient hands of centuries and centuries of workers, some of them nameless and unknown who without thought of reward, unheralded and unsung, played their part and did the day's work. To all these we pay tribute. They, with others, laid the foundation on which we build.

CONCLUSION

No one can study the history of periodontia and not be impressed by the fact that, like everything else in life, it has been subject to the law of evolution. As it emerges from the obscurity of the past it is seen to be enveloped in almost complete ignorance. It had been observed that as a result of disease affecting the periodontal tissues the teeth became loosened and eventually were lost. Why this was so, no one seemed to know and few were interested enough to seek the answer. Attempts at treatment were made, but as might be expected, these were empirical and usually of little value. It was not until after the birth of American dentistry that any real progress was made, so slow is the growth of knowledge. It is probably not too much to say that the most potent influence in the recent progress of periodontia was the formation, twenty-five years ago, of the American Academy of Periodontology. Too much credit can not be given to Drs. Gillette Hayden and Grace Rogers Spalding. To them more than to anyone else in the profession, we are indebted for the present status of periodontia. It was largely due to their vision and perseverance that the effort was made to bring together in one organization those known to have an interest in the subject.[54] Today we celebrate that event—the most important in the evolution of periodontia. New life was at once infused into the subject. Order was brought out of chaos. A new nomenclature was created. Attempts at classification of periodontal diseases were made. Articles and books were written covering many phases of the subject. In an effort to aid the general practitioner a section of periodontia was organized in the American Dental Association. Articles were written and lectures given for the instruction of the family dentist and the laity. In many ways and along many lines the Academy has gone forward in its campaign of education. Few organizations have contributed more in the past quarter of a century toward the progress of dentistry. It has more than justified its existence.

But as it stands on the threshold of another quarter century it will be seen that its work is not yet finished—indeed has hardly begun. As it faces the future, it finds that more teeth are lost from periodontoclasia than from any other single cause despite all the progress that has been made since the days of Riggs.[55] It finds, too, that there are at present probably fifty thousand dentists in the United States who have been given little or no undergraduate instruction in periodontia, who have been graduated and licensed to practice in almost complete ignorance of one of the most fundamental branches of dentistry. It is not surprising, therefore, that many dentists are still telling their patients that "pyorrhea is incurable." Nor is it difficult to understand why it is that periodontal diseases are almost universal, even among those who are regular patrons of the family dentist.

And lastly, it will be found, as a survey is made of present day dental education, that periodontia is still far from receiving the attention from dental educators which its importance deserves. In a recent report along these lines it was stated "that the evaluation of periodontia in the average undergraduate curriculum is far from commensurate with the importance of the subject. In ten of the catalogues (studied)

there was little or no mention of periodontology either in the faculty or the schedule, or the description of the course of study."[33] This is unfortunate for it is only through proper training of the undergraduate student in dentistry that the problem of periodontia will ever be solved. It cannot be done by a small group of specialists no matter how progressive they may be. Another quarter century, however, should see many changes for the better along all these and other lines.

Provision must also be made for the post graduate instruction and certification of the specialist in periodontia. This is highly important, for there are at present altogether too many so-called specialists whose knowledge of the principles and practice of periodontia is far from adequate. This situation cannot be corrected by a two weeks post graduate course of instruction helpful though that may be as a temporary measure. Fortunately the Academy is alive to its responsibility along these lines and is already engaged in formulating plans for the training and certification of specialists in periodontia. These must provide a much broader foundation than that comprehended in our present conception of the science and art of periodontology, a fact fully realized by those having the matter in charge.

And so, in the light of what has been accomplished in the past, and visioning the needs of the future, who can doubt that another quarter of a century will see even greater progress in the science and practice of periodontology for is it not true as has been said, that

"Progress, is man's distinctive mark alone,

Not God's, and not the beast's; God is, they are;

Man partly is, and wholly hopes to be."

580 FIFTH AVENUE

REFERENCES

1. Weinberger, B. W.: *American Historical Review of Orthodontics.* 1:p.138.
2. *Ibid:* p.157; Guerini, Vincenzo: *History of Dentistry.* Philadelphia, Lea and Febiger. 1909. p.318.
3. Hulin, Ch.: Historical & Experimental Study. *L'Odontologie,* 73(2):73. Feb., 1935.
4. Guerini: p.309. (see Guerini, 2 above).
5. *Ibid:* p.330.
6. Prinz, Hermann: *Diseases of the Soft Structures of the Teeth,* p.343.
7. Personal letter to the author by Dr. Balint Orban.
8. *D. Cosmos,* 27:766. Dec., 1885.
9. *Ibid,* 24:527. Oct., 1882.
10. *Johnston's Dental Miscellany,* Aug., 1878. p.306.
11. Letter in possession of Dr. James F. Hasbrouck.
12. Personal letter from the Dean of the Baltimore College of Dental Surgery.
13. Thorpe, Burton L.: *History of Dental Surgery.* 3:346-356.
14. *D. Cosmos.* 19:572. Nov., 1877.
15. *Ibid,* 31:562. July, 1889.
16. Hulin, (see 3 above).
17. Black, G. V.: *Special Dental Pathology.* p.67.
18. *D. Cosmos,* 57:1193. Oct., 1915.
19. Black: p.50 (see 17 above).
20. *D. Cosmos,* 62:1365. 1920.
21. *Ibid,* 36:727. 1894.
22. *Ibid,* 38:676. Aug., 1896; *J. D. Res.,* 3:154. Dec., 1921.
23. *Ibid,* 62:911. July, 1920.
24. *D. Summary.* June, 1909.
25. Six Years' Work in Oral Prophylaxis. Reprint (origin not given).
26. *D. Cosmos,* 64:797. July, 1922 & 78:12. Jan., 1936.
27. Thorpe: p.613 (see 13 above).
28. *D. Cosmos,* 67:216. Feb., 1925.
29. Talbot, E. S.: *Interstitial Gingivitis, or so-called Pyorrhea Alveolaris.* 1899. p.174.
30. *D. Cosmos,* 27:669. Nov., 1885.
31. *Ibid,* 30:798. Nov., 1888.
32. *J. A. D. A.,* 4:580. June, 1917.
33. *D. Cosmos,* 72:312. Mar., 1930.

34. *Ibid*, 76:389. Mar., 1934.
35. *D. Summary*, Reprint, no date (probably 1908).
36. *D. Cosmos*, 56:642. May, 1914.
37. *J. A. D. A.*, 20:528. Mar., 1933.
38. *D. Cosmos*, 64:555. July, 1922.
39. *J. Periodont.*, 3:100. Jan., 1932.
40. *J. A. D. A.*, 18:2210. Nov., 1931.
41. *Ibid.*, 7:155. Feb., 1920.
42. *J. Periodont.*, 5:58. Jan., 1933.
43. *J. A. D. A.*, 25:798. May, 1938; *Ann. Den.*, p.127. June, 1938.
44. *J. Periodont.*, 7:37. Jan., 1936.
45. *Ibid.*, 1:13. Jan., 1930.
46 *D. Cosmos*, 71:735. July, 1929.
47. *J. Periodont.*, 1:11-12. Jan., 1930.
48. *J. A. D. A.*, 16:924. May, 1929.
49. *D. Cosmos*, 13:17. Jan., 1871; 40:233. Mar., 1898.
50. Harlan, A. W.: *American System of Dentistry*, Philadelphia, Lea Brothers, 1887. pp.273-92.
51. *D. Cosmos*, 19:70. Feb., 1877.
52. *Ibid*, 21:192. Apr., 1879.
53. *Ibid*, 36:779. Oct., 1894; *J. A. D. A.*, 4:521. May, 1917.
54. History of the A. A. P., *Ann. Den.*, p.28. Mar., 1937.
55. *D. Cosmos*, 71:2247. Dec., 1929.
56. The Status of Periodontia in the Dental College Curriculum, *J. A. D. A.*, 23:1079. June, 1936.
57. *D. Cosmos*, 72:675. June, 1930.

PROGRESS IN THE RECOGNITION OF ETIOLOGIC FACTORS OF PERIODONTAL DISEASES*

By A. W. BRYAN, D. D. S., F. A. C. D.
Iowa City, Iowa

IN A search for historical data having to do with diseases of man, one is impressed with the fact that many of those afflictions which we are inclined to consider as relatively modern are, in fact, probably as old as man himself. Diseases of the supporting structures of the teeth are no exception, and many historical books refer to the evidences found in the skulls of the earliest cave-dwellers showing extensive deposits upon the roots of the teeth and destruction of alveolar tissues that undoubtedly corresponded to the conditions we know today as some type of dental periclasia.

In a search for historical data on a subject of this nature one is handicapped by not having access to original material. It is, therefore, necessary to depend upon articles and books written by others who have been fortunate in having made contacts with archaeologists or with the publications containing their findings.

The majority of us who may be interested in delving into the history of gingival disease are limited to the literature as found in the average college or university library. Acknowledgment should be made for the outstanding contribution that has been made to dentistry and medicine in the development during the past few years of the *Index—Dental Periodical Literature*. Needless to say that the late Arthur D. Black, in addition to many other important contributions to dental science, built a monument to himself in the part he played in this work. Previous to the time when the Index was available, the search for an article on any subject in dentistry meant hours of tedious labor without guidance and no assurance of success in gaining the information desired. With this Index now at hand, reference to any article published in recognized dental periodicals since 1839 may be quickly found.

Those of you who have used the Index know that it is classified to follow the various topics to be found in the study and practice of dentistry. Under the division of "Diseases of Gingivae and Peridental Membrane" it is, therefore, easy to find all articles published in eighty-three periodicals in the United States, England, and Canada since 1839.

*Read before the American Academy of Periodontology, October 20, 1938; St. Louis, Mo.

In an examination of the section just referred to, it was found that there are more than 5,700 articles listed on the diseases that would be included under "dental periclasia" as this Academy accepts that term. Obviously it would be the work of months or years to even scan these articles to obtain the gist of the author's message. Articles in this group in which the title had specifically to do with "etiology" numbered more than 400. Review of these alone would be a long task and doubtless nearly every article listed deals to some extent with etiology even though the title refers to treatment or some other phase of the condition.

It is interesting to note that the articles on etiology of so-called pyorrhea seem to come in waves or surges. As a new theory is brought out the literature soon is filled with articles dealing with the new long-sought cause, with, of course, approval and disapproval divided among the writers.

Considering for a moment some of the early references to diseases of the periodontal tissues, we find the Ebers Papyrus[3] which, according to George Ebers, a German Egyptologist, was probably written some 1600 years before the Christian era, or perhaps even to 37 centuries B. C. In it mention is made of preparations which may be used to "strengthen the gums." It seems evident that, from the nature of the remedies prescribed in these earliest of manuscripts, the etiology was looked upon as a matter of uncleanliness and the accumulation of irritating materials. Fauchard[4] in 1723 remarked that lack of cleanliness and care was ordinarily the cause of the maladies which destroyed the teeth.

In 1778 M. Jourdain[5] discussed the disease from the standpoint of possible relationship to scurvy,—interesting in the light of recent articles based upon the effect of dietary deficiencies upon the periodontal tissues. There seem to be few references.

In 1860, Marshall de Calve[6] advanced the opinion that heredity played a part as a cause of pyorrhea. In the use of the term pyorrhea it is of interest to note that there seems to be a general belief that it was first used by Riggs. However, in Eugene S. Talbot's book "Interstitial Gingivitis"[14] he states that in 1822 M. Joirac[6] referred to the disease under discussion as "pyorrhea inter-alveolo-dentaire." According to Black,[13] the term "pyorrhea alveolaris" was introduced by F. H. Rehwinkel at the meeting of the American Dental Association in 1877.* Talbot also refers to an article by Magitot in 1867 (without naming the publication), in which he states it as his opinion that systemic disorders such as gout, rheumatism, albuminuria, diabetes, and anemia should be considered in the search for causes. Bonwill[7] in 1867 offered an opinion that the alveolar support was inadequate through thinness of the septal bone, and therefore the peridental membrane and gingiva were destroyed. He also mentioned the want of proper articulation of the teeth as a factor. This is of interest as it relates to the importance of traumatic occlusion, a factor now given much attention in our consideration of etiology.

In 1875 Scheff[8] of Vienna stated that he believed pyorrhea originated from external causes such as irritation from mechanical, thermal, and chemical changes.

In 1876 an Italian investigator, Sirelli,[9] advanced the opinion that rheumatism, scrofula, syphilis, and other constitutional diseases might be responsible along with local irritations as exciting factors.

In 1877 Rehwinkel[10] laid stress on acquired and inherited constitutional defects and thought that salivary calculus was of questionable importance. Clowes[11] was a believer in a systemic relationship from the nutritional aspect. On the other hand, there were many investigators in this same period who did not believe that constitutional conditions were related to the etiology. Niles[12] expressed the opinion that they were not frequently of any importance. However, he believed that excessive intake of lime salts in drinking water had some effect and it is difficult to see how he was able to divorce this action entirely from some constitutional process.

Since John M. Riggs is so closely linked with the history of diseases of the periodontium, some mention of the part which he played should be made at this time. Merritt[6] in a paper read before this Academy in 1921 stated that Riggs had written only two articles on the subject and that only one of these was published in full. As is generally known, many of the profession used the term "Riggs disease" in place of "Pyorrhea alveolaris" and he (Dr. Riggs), was subjected to some criticism by the profession for thus allowing his name to be used. Obviously he could have had no control over

*Probably first used in America by Dr. Rehwinkel—ED.

such a matter and publicly disclaimed having ever used the term himself or of originating it. Riggs defined the disease as "suppurative inflammation and absorption of the gums" or "scurvy of the gums." Even though the term "scurvy" might indicate, however, a systemic relationship as to cause, he believed the causes generally to be of local character. Merritt[6] refers to the fact that Riggs practiced and taught the surgical treatment of the disease. He decried the use of drugs in its treatment except to relieve pain and soreness immediately after the operation. In an article, "Pyorrhea Alveolaris,"[14] he took exception to the statements of other writers who held that "the looseness of the teeth which characterizes this disease arises from a lack of nerve energy." He went on to cite cases in his own practice in which the vigor of the patients disproved these claims. He did state, however, that "it is not lack of nervous energy that causes this disease, but the disease will lead to loss of nerve energy." It is evident then that Riggs did not consider constitutional factors as of much importance in etiology.

Mention was made in the beginning of the tides or waves of papers that were written on the subject concerned in this paper. The period from 1881 to 1885 seems to represent one of these periods. Several papers appeared which laid much stress on the bacteriologic phases of the etiology. It seems that from that time even to the present there have been numerous investigators who have considered the matter of infection as one of great importance. This is borne out by the fact that many of the treatments that have been advocated up to the last few years have been based upon antiseptic or germicidal action. The whole theory of the treatment by use of serums is of course predicated on the infective factor.

In 1894 G. V. Black[15] presented an article on Diseases of the Peridental Membrane and the Uric Acid Diathesis in which he discussed a paper by C. N. Pierce, who had expressed the view that the disease under discussion did not start at the gingival margin, the generally accepted belief as regards all cases of pyorrhea at that time, but that it began on the sides of the root, most likely near the apex. He held that it extended from this point, separating the membranes from the tooth, finally opening at the gum margin with the formation of pus pockets. He connected the disease definitely with gouty diathesis as the underlying cause and stated that the presence of uric acid in the concretions found upon the roots of the teeth demonstrated proof of the connection. In passing, it is of interest to note that the later researches of Box deal with a phase of dental periclasia which he terms "rarefying pericementitis fibrosa," quite similar in its beginning pathology to that described by Pierce. However, as we all know, the theories as to etiology are widely different. Black discussed Pierce's paper and, after many chemical tests for the presence of uric acid in calculus, declared that there was no essential difference in the content of that substance in patients with and without gout. He concluded that after a close review of all the facts known about the disease, called by him "phagadenic pericementitis," there was a "strong balance in favor of the idea that it was a purely local affection of an infectious character that has relation to a particular tissue,—the glands of the peridental membrane."

Mention has been previously made of the stress placed upon the bacterial phase of etiology. This theory had many advocates and numerous articles that have been written on treatment have been based on that belief. In the period around 1912 to 1916 quite a flurry in the use of vaccines for treatment occurred. This treatment was of course based on the premise that infective organisms caused the disease. Even today there are many supporters of the infective theory. Hartzell[16], the author of many articles on the subject, considers streptococcic infection as one of the important etiologic factors. In an article in 1935[17] he states, "Certain it is in my mind that if the dental profession of the world would utilize the principles demonstrated by Pasteur and Duclaux and accept the fact that periodontoclasia or pyorrhea alveolaris is purely and simply a germ ferment disease and that the destruction of the periodontal membrane and the bone surrounding it and the tissues surrounding the bone is due to the action of pus-making bacterial ferment, enormous progress would be made, and dentistry as a learned profession would have received a practical uplift in value and in the minds of the medical profession, who have viewed our operations largely as mechanical."

In 1914 two groups of investigators

working independently reported investigations on the presence of certain types of protozoa in the mouth in cases of pyorrhea. Barrett,[16] working with A. J. Smith in the University of Pennsylvania, found that parasitic amoeba in active motility were present in many cases of suppurative affections of the gingivae. Assuming that the presence of these organisms accounted at least in part for the disease treatment with emetin, a recognized amoebicide, was recommended. The other investigators were Bass and Johns of St. Louis and similar work was done by them. The older members of this group remember the enthusiasm with which this theory was received. The end-result was a tremendous increase in sales of emetin and the development of many sore arms from the injection of said drug by amateurs in the use of the hypodermic.

One of the most important contributions to the knowledge of etiology of dental periclasia has to do with the relationship of occlusion. Merritt[8] calls attention to one of the earliest articles on this subject by Quedenfeldt in 1908 and remarks that little attention was paid to his teachings at that time. But in 1917 Stillman[18] wrote on the possibility of damage being done to supporting tissues through inharmonious, inclined-plane relationships and the production of excessive stresses on these tissues. The damage came not so much from stress delivered at normal angles to the long axis of the tooth but because of force in abnormal directions upon the peridental membrane and alveolar bone. There is some debate on the question as to whether so-called traumatic occlusion is not, in many cases, the result of certain types of periclasia rather than the cause; but clinical results which are obtained when it is carefully considered in treatment, justify the belief of many operators that it is often an important etiologic factor. For instance, the balancing of stress in the region of the incisors no doubt prevents the destruction of alveolar support in many cases, and grinding of these teeth is justified even though no beginning pathology is apparent. Box[19] of Toronto has, in numerous articles, pointed out the effects of excessive stresses on the peridental membrane and the alveolus. In the condition which he calls "rarefying pericementitis fibrosa" he attributes the characteristic changes to stress, and supports his contention by pointing out that the changes begin in those areas subjected to the severest strain.

It is not possible to make reference to all of the numerous articles that have appeared for and against the theory of traumatic occlusion. Probably the author who has minimized its importance and effect the most is H. W. MacMillan who maintains that the periodontal tissues are so formed and adjusted that they withstand all stresses of mastication without injury. Articles supporting the theory have appeared by J. O. McCall, Austin F. James, Sidney Sorrin, and many others.

One of the most discussed subjects in relation to etiology of dental periclasia is that of nutrition. Dentistry became tremendously interested in the subject of diet in the years following 1918. The work of Percy Howe, while primarily directed toward research in dental caries, yielded much valuable information on the effects of dietary deficiencies on the condition of the tissues which support the teeth. Only three of his articles[20] are listed as reference in this paper, and they are of relatively recent date. However, he has written many papers in which reference is made to this phase of the subject. Howe has demonstrated that changes in the alveolar bone of experimental animals may be produced at will by administering diets which are deficient in various essential elements. His feeding experiments with guinea pigs and rats, in which, with a scurvy-producing diet, he is able to cause rapid resorption of the alveolar support, are familiar to all of us. These experiments are easy to reproduce and have been by many experimenters. We have specimens in our possession in which the inferior border of the mandibles are completely honeycombed after three weeks of feeding with this diet. Howe has demonstrated also that at any time during the progress of this resorptive process it is possible to cause a deposition of calcium and phosphorus and repair of the damage which has occurred.

The question has been raised with Howe as to the reason for the effects upon the osseous structures with a vitamin C deficiency when such deficiencies usually result more in soft tissue changes. It was pointed out at the same time that the diet employed seemed adequate in minerals. His answer to the question was that in certain laboratory animals there was a profound effect upon

the whole intestinal tract and, in the case of the guinea pig especially, constipation was produced. Several other factors then enter the picture. Metabolism is interfered with and even though adequate mineral supply is present in the food, its absorption and utilization is prevented. Also, the factor of absorption of toxic products from the inactive bowel must be considered. One is led to raise the question as to the importance of this factor in many of the pathological conditions found in the mouth. Is it not possible that the burden of elimination of toxins thrown upon the system may affect the whole calcium balance in the blood? Vines[21] has stated that toxins are eliminated by combining with the ionic calcium of the blood, thus lowering the blood levels of that mineral with a call upon the storehouse (bones) for replenishment.

In addition to the effect of nutritional deficiencies in a purely local sense, such as removal of lime salts or the production of capillary engorgement, we must consider the systemic well-being and its reflection in the local resistance of the tissues with which we are concerned.

Many periodontists do not consider that nutrition is a very serious etiological factor in periclasia, but it seems to the writer that the general trend, as seen in present day literature, indicates an increasing acceptance of its importance. In 1922, "A Textbook of Clinical Periodontia" by Stillman and McCall was published. In it was a comprehensive chart on etiology. Among those causes listed as secondary was "dietary influence." We wrote to the authors for permission to reproduce the chart for teaching purposes; they replied that it was in the process of revision and that it would be well to wait until it was complete. We made a prediction to the students that one of the changes would be to assign this factor to a more important place in the chart. Our prediction proved true and in the new edition "dietary influence" was listed under both primary and secondary divisions.

While there has been no attempt to make a complete historical record in this paper mention should be made of the contributions of Clarence J. Grieves. His work was along the lines pursued by Percy Howe and substantiated in large measure the conclusions drawn by him.

In closing let us discuss briefly the present day concepts of the etiology of periclasia. We must admit that there is still much difference of opinion. It has not been possible to single out one specific cause for any or all of the types of periclasia. Health in any tissue seems to be a matter of resistance balanced against severity or virulence of the irritant. Certain it is that the majority of periodontists no longer think of a single factor being the cause. We have discovered that we cannot set the oral cavity off by itself as a unit, separating it from the body, and disregarding all of the metabolic processes that influence general health as well as local health. Mechanical irritation and trauma in the local area are present in practically all cases of periclasia, but we have all seen cases with severe local irritation and the only result observable over a long period of time was a moderately severe gingivitis. Why the difference? The same *local* etiologic factors are there but the results are different. Weston A. Price has pointed out that individuals vary greatly in the reaction of tissues against irritants, mechanical or infectious, and these differences are clearly seen in the cases mentioned above. While it was not the intention to deal in this paper with the subject of treatment, it seems desirable to emphasize the fact that rational therapeutics of *any* disease is based upon removal of the cause. Hence, the first and most important step in every case is to determine those factors responsible. If this is not done, we resort to treatment of symptoms. It is true of course that symptoms must be treated but, if the treatment of the case is confined wholly to that, relief is only temporary, for we have allowed the original causative factors to remain. It seems to the writer that drug treatment, unless directed at specific infection, and the surgical removal of unattached tissue are both essentially the treatment of symptoms. Unless the deep conditions that caused the unattached tissue to be formed or allowed the infection to become virulent are corrected, we have not treated our case scientifically.

I believe that the profession can justly claim much progress in the recognition of etiologic factors of periodontal disease, and perhaps the greatest progress is indicated in the recognition of the oral tissues as an inseparable part of a complete organism.

REFERENCES

1. Guerini, Vincenzo: *History of Dentistry*, Philadelphia, Lea & Febiger. 1909.
2. Jourdain, M.: *Philadelphia J. M. and Physical Sc.* 1821.
3. De Calve, Marshall: *J. A. M. A.* (Vol. not indicated.)
4. Talbot, Eugene S.: *Interstitial Gingivitis or So-Called Pyorrhea Alveolaris*, S. S. White Dental Mfg. Co. 1899.
5. Joirac, M.: *J. A. M. A.* (Vol. not indicated.)
6. Merritt, A. H.: A Brief History of Periodontology. *J. D. Res.* 3(4): 149-59. Dec., 1921.
7. Bonwill, W. G.: *D. Cosmos* 1884.
8. Scheff. *Wiener Medical Presse.* 14. Vienna.
9. Sirelli: *Gazetta Medical di Roma*, 1876.
10. Rehwinkle, F. H.: Pyorrhea Alveolaris. *D. Cosmos* 19:572-74. Nov., 1877.
11. Clowes, J. W.: *D. Cosmos* 21. 1879.
12. Niles, N. S.: Phosphoric Acids, Zinc Phosphates, and The New Metal Actinium. *D. Cosmos* 24: 245. May, 1882.
13. Black, G. V.: Diseases of the Peridental Membrane and the Uric Acid Diathesis. *D. Review* 8:449. 1894.
14. Riggs, J. M.: Pyorrhea Alveolaris. *D. Cosmos* 24: 524. Oct., 1882.
15. Hartzell, T. B. and Henrici, A. T.: The Pathogenicity of Mouth Streptococci and Their Role in the Etiology of Dental Diseases. *J. A. D. A.* 4(5): 477. 1917.
16. Barrett, M. T.: The Protozoa of the Mouth in Relation to Pyorrhea Alveolaris. *J. N. D. A.*, 1914.
17. Hartzell, T. B.: The Story of the Origin and Development of Pyorrhea. *J. A. D. A.* 1935.
18. Stillman, Paul R.: The Management of Pyorrhea. *D. Cosmos*, 59: 405. 1917.
19. Box, Harold K.: Experimental Traumatogenic Occlusion in Sheep. *Oral Health.* 1935.
20. Howe, Percy: Diet as a Fundamental in Dental Conditions. *J. A. D. A.* 15: 1673. Sept., 1928. Nutritional Aspects of Dental Disease. *Oral Topics*, p. 571. June, 1932.
21. Vines, H. W. C.: *The Parathyroid Glands in Relation to Disease.* London, Edward Arnold Co. 1924.

ATTENTION!

Members of the American and Canadian Dental Associations are invited to attend the Twenty-Sixth Annual Meeting of the American Academy of Periodontology, to be held in Milwaukee, Wisconsin, July 13, 14, 15, 1939. See pages 47 and 48 of this Journal for complete program.

RAYMOND E. JOHNSON, *Secretary*

THE PAST AND FUTURE OF PERIODONTIA*

By PAUL R. STILLMAN, D. D. S., F. A. C. D., F. A. A. P., *Longwood, Florida*

IT SHOULD offend no one if the author of this paper discusses his own ideas and mental processes. Parenthetically, these have changed considerably during the quarter of a century since this Academy was founded, as any opinion is subject to change: Some of the charter members will remember the first Annual Meeting. Those who met then were dentists who had shown an interest in, and had presumably developed some skill in the treatment of "pyorrhea alveolaris." They had been called together for the purpose of founding an organization for study and for the advancement of the biological arts and sciences of dentistry, as these applied to what we now call periodontal disease. How well that idea has succeeded each member may ask and answer personally. To this writer, the Academy has provided a stimulating contact with others, who like himself, were interested in the subject.

As the years have passed, we have seen ideas and theories come and go. The suitable has been retained and the unsuitable has been passed. Not all, however, for this takes time—years. In the development of a profession, an idea apparently requires several generations of human life in which to mature. When an idea is in its infancy, hope is misled by imagination, and complacency reigns. In the early infancy of Periodontology, as it developed under the ideologic traditions, which the Academy members inherited from its parent subject Dentistry, interest turned to the subject of the d e s i g n of instruments,—"scalers." Differences of opinion arose. Interest developed heat. And for a short while there was danger that two schools, or sects, would develop schismatical differences within the young organization.

The idea of technique is applicable to technology and that subject dominated the interests of all dentists, including the periodontists of the day. The better instrument represented the idea of a better means for a c c o m p l i s h i n g the purpose—and Periodontia had adopted a purpose,—*e. g.* the treatment (and cure) of pyorrhea. The term "pyorrhea" had been accepted in dentistry to designate the name of a disease, the most obvious objective symptom of which was pus flow. Treatment of the disease consisted largely of detaching accumulations of so-called "serumal deposits" found upon the roots of the teeth. In the mind of far too many dentists, removal of these deposits still constitutes the sum total of the treatment of periodontal disease. The deposits were identified as "causative factors" of pus flow, a symptom which identified the disease. "Remove the cause" is a sound idea—But *how* shall the cause be removed was the question.

Naturally the design of steel instruments became a subject of controversy. The seriousness with which the subject was discussed may be reviewed today as trivial, but the design of scalers was very seriously discussed in the earlier days of this Academy, and rightly so, presupposing the scaler as a means to an end. Our former preoccupation with scalers may be likened to the situation that would exist if authors were to argue over the type of writing instrument each preferred. In literary art that *is* trivial.

The next step in periodontology had to do with nomenclature. The term "pyorrhea" was euphoniously objectionable. The so-called "pyorrhea specialists" did not like this name. A nomenclature committee was appointed to solve an embarrassing problem. Many of their recommendations were adopted. The pyorrhea specialist became a "periodontist" and that term is now popular. But it was a change of name, not one of immediate change of ideas. Interests change easily in youth, but when an idea is once accepted it is tenaciously held. Any unfamiliar idea is not easily accepted, regardless of its obvious soundness and validity. The process requires a displacement of the idea held, before an unfamiliar idea can be accepted.

But let us go back a little. Before dentistry became a profession it existed as a trade. I do not mean to imply that dentistry was exclusively a trade, that there were no truly "professional" men in dentistry in earlier days. Here and there outstanding

*Read before the American Academy of Periodontology October 21, 1938 at St. Louis, Missouri.

names appear on the pages of dental history; incidentally, those leaders, almost without exception, had more than a smattering of medical education and hence possessed the biological viewpoint. But by and large dentistry was philosophically a trade. And while it was a trade it developed an ideology. The trade was an art—a technological art. Dentistry became a profession under the influence of a stated formula,—"dentistry is a profession." Under that idea, thought turned to education. A new system of education was adopted, with the founding of a dental college. That event occurred in 1840. The knowledges of dentistry and the ideology of the dentists were adopted by the college.

About three quarters of a century of professional development elapsed before the idea of periodontology appeared, and that also was an event. The founders of the Academy brought to it a philosophy of technological dentistry but the idea was not recognized as such at the time. The controversy over the design of instruments, current at that time, reveals the nature of the interest. But were we not dentists?

And now for the problem of periodontal disease. Disease is clearly a biological problem. Pyorrhea, comprehended as pus flow, is a biological phenomenon. The technological interests of the dentists had developed an objective method of thought and the disease pyorrhea was "seen" and accepted as *an object*, whereas it could only be comprehended as *a subject*. That idea identifies a major intellectual difficulty at this present time. The differentiation between an object and a subject is really very simple, as soon as the mind consents to the idea. But it is difficult indeed for a mind to consent. It requires reflection.

The term periodontology identifies a subject, while "pyorrhea" identified an object. Subjects permit thought and here ideas proceed to understanding. An object may promote "wonder," but wonder does not identify thought. It can promote curiosity but wonder is always mixed with "a feeling of doubt." But an idea may easily become side-tracked. One interest can supersede another. The early interest in the design of instruments was dropped before it resulted in injury to the Academy. The subject of nomenclature superseded it. This new interest produced satisfaction and the Academy members recognized it as a constructive intellectual movement.

Then interest turned to pathology,—*dental* pathology. It was recognized as a science subject. The subject was pursued largely through the medium of histology. Microphotographs were produced and dentists were permitted to "see" objects, viz.—photographs showing tissue changes. These they could comprehend, for dentists can think through their eyesight. Undoubtedly, consent would have been given to the idea that dental pathology is a biological subject and also that a biological subject is prefaced by the subject of life present. There is no life present in anatomy, none in a photograph either, even when the subject pertains to life. The presentation of histopathology can be comprehended as minute anatomy and that is comprehensible under the objective method of thought—but that is not pathology.

Pathology is defined as, "The branch of medical science that treats of a morbid condition of an organism." We call it disease, but an organism differs in an important principle from a cadaver—for a cadaver is devoid of life-present. Thus the idea can be comprehended that disease cannot exist apart from the idea of life present in an organism. The cadaveric material studied as pathology and interpreted by microphotography is indeed a valuable basis for study but it should not be mistaken for a study of disease.

What then is disease? Can it by any chance be photographed? It would be wise to accept the *Standard Dictionary* definition of disease and compare that with the ideas presented as *dental* pathology—"*Disease*—an incoördination or disbalance of physiological action." That idea cannot be grasped by an objective method of thought. Disease is a subject. A lesion can be photographed and this photographic presentation can provoke thought, but it cannot present disease. What then is "physiological action?" It is biological function or *vitae phenomina*. Physiology is properly a medical science, devoted wholly to the idea of vital function, but as yet it has no counterpart in the literature of dental science. Dental pathology as it now exists is *nominal* pathology; an euphemistic delusion. It is anatomical and morphological, but not yet

biological. When dentistry possesses dental physiology, dental pathology will arrive at its adolescence.

In the latest edition of *Encyclopedia Britannia* there is an article explaining what pathology is, and how it should be understood. To this writer, that article revealed his own misconception of pathology, which had been derived from "dental pathology." Many unfamiliar ideas and principles are presented there. The fact was revealed that pathology is not comprehensible without a knowledge of physiology—and dental physiology has not yet appeared as a published volume. And, a personal opinion is here expressed that biological thought in dentistry is not yet sufficiently developed to produce a volume of Dental Physiology. Dentistry is youth-like, but no youth cares to admit or be told "You are too young to know." Wisdom lies in knowing that one *does not* know. "Wisdom is mental power acting upon the materials that fullest knowledge gives in the most effective way." Not all material is matter; *e.g.* literary material.

Following the idea of disease, there came to dentistry and through periodontology, an idea as to its true function as an art. A biological idea had been presented to dentistry as a formula. It read "Dentistry is a branch of the healing art, coequal to a specialty of medicine." Medicine is defined as "the healing art"—a biological art. The young and very immature specialty of dentistry, which we have named periodontia, has now opened up an ideological pathway which is also biological. Periodontists however, are dentists, their problem is biological only, while dentistry's problem remains technological—prosthetic.

All of dentistry, including the art of periodontia, is faced with a philosophical problem. A biological art and a technological art present irreconcilable fundamental differences in principles. These two arts can never meet upon a common ground of understanding. Both are sound and right so long as they remain separated and in their own intellectual habitat.

No one really loves or respects a mule. Even nature abhors it. For a mule is neither horse nor ass. An ass is an humble beast, while a horse is ideologically a steed. The differences in dentistry exist in the idea of art, not in science. Art is something to be done; science is something to be known. That statement identifies the first and present cause of confusion. The technological art of dentistry can be observed (subjectively) as dental prosthesis. That is a subject capable of definition, for it presents a definite principle.

The general principle of prosthesis is "The making of artificial parts and fitting them to the living human body." Examples falling within the special interests of surgery are artificial eyes, legs, etc. But of equal interest to human welfare is the principle of prosthesis as it is applied in dentistry. Mechanical dentistry was prosthetic in principle before the term "mechanical dentistry" was abandoned; and "operative dentistry" is today prosthetic in principle. And all prosthesis is technological, including a gold-foil filling and similar restorations. A "cavity" is an object and filling a cavity is prosthetic in principle. When the cavity has been filled it is no longer a cavity. But the scheme of filling was successful in so far as checking the further progress of the carious process is concerned. Only an artisan could have conceived the idea. Medicine is opposed to empiricism, especially when it runs counter to the philosophical principles of the healing art, and filling teeth does that.

I have said that artificial eyes and legs are examples of prosthesis, and it may be inferred that I put on a plane with them the bridges and dentures in which dentists take such pride. Whether this is, or is not true, depends *not* on the bridge or denture itself, but on the conception of the dentist who constructed it. Often the dental appliance is constructed without any conception of the physiology of the teeth and in such cases it descends to the level of the glass eye, satisfying, after a fashion, the esthetic demands of the case, but serving no other useful purpose. In fact, such "pieces" frequently are of a lower order than the glass eye or artificial limb, in that they induce irritation and disease. On the other hand, dental prosthesis, if properly conceived, will not only *not* cause irritation but may be a means of assisting in the recovery of the mouth from a condition of disease. It then becomes one of the agencies that may be invoked by the dentist—a health service.

Let me return, however, to our main theme. The mental struggle of dentistry to arrive at its present state of development began before the formula "dentistry is a profession" appeared. A vague hope is a vain hope—akin to wonder. But when an idea is formulated, a means for action and expression becomes available. The formula "dentistry is a profession" developed from the idea that a relation existed between medicine and dentistry, and that idea holds —and *will* hold, as a *service* but *not* as an art. That formula turned the attention of dentists to the subject of education and away from their greatest desire.

A profession requires that its members be educated men and women, and very little "liberal education" existed among the artisan-dentists prior to the beginning of dentistry's scholastic era. The knowledges of dentistry as these then existed, under the indenture system of education, were adopted as "the equivalent" of liberal education. That idea satisfied dentists, only. But the greater difficulty of the founders of the first college was the objective method of thought, which was universal, especially among dentists. The names of objects were adopted to represent subjects to be taught in the *new* college and that was a serious handicap for scholastic education. Dentistry is now comprehensible as a subject, regardless of its characteristics, but the terms "operative dentistry" and "mechanical dentistry" led thought to objects and these objects are traditions of dental education as it now exists. To question the propriety of established custom is iconoclastic; an image breaker is never popular, especially when the image is a respected traditional belief. Nevertheless, if one desires to see light, even mental light, iconoclasm is more efficacious than mental darkness.

The first formula, "dentistry is a profession," transformed dentistry from a trade—*fait accompli.* Dentistry's second formula—"dentistry is a branch of the healing art, coequal to a specialty of medicine," has already produced many new ideas, and its influence upon the Academy of Periodontology will result in the acceptance of new principles for periodontia. But new and unfamiliar ideas frequently lead to tangents. That was the case with the first formula. It is here suggested that interests of the present remain, for a while at least, upon academically accepted principles. The art of periodontia can easily accept the idea that it *is* "healing art"; and that *is* biological art, as the term "healing" implies. That idea will provide a distinction between the principle of prosthesis, which is technological, and healing, which is purely biological, and will serve to distinguish periodontal treatment, which is biological, from replacement of missing parts or units, which is technological. The fact that restorations are frequently needed in the course of periodontal treatment should not and need not cause confusion.

Recently, dentistry has taken an active interest in biological science. Many dentists are working in the field of science, but we should remember that dentistry is an art. The releases of research workers are biological only to the extent to which they have retained the idea of *life present in material.* The interests of periodontia lie in art, as art relates to the idea of healing, the product of which is health—oral health. Health cannot appear in the principle of prosthesis, for that is technological, and health is biological. Two incompatible philosophies are recognized. Both are art, but while one cannot be translated into the other, both may be utilized, either alone or in combination, in case management. It is obvious, however, that health cannot be fabricated, for fabrication implies inert material, and on this idea the principle of technological art is based.

The term philosophy represents to most minds a very abstruse and abstract idea. It is essentially a discussion of general principles, and dentistry possesses general principles. Let us then, give thought to the accepted principles of our profession and when we do that, we have entered dental philosophy. Ideology does not penetrate far enough into the subject of general principles to be particularly useful at present. First, let us not confuse science with art. Both are subjects which present separate perspectives of a single interest, but these "things" are interdependent and philosophy lies between the two. Science represents something to be known and the true scientists are not concerned with art. They are concerned only with the subjects *truth* and *fact.* If the knowledge they develop is pertinent to art, they are pleased. If it is not immediately useful to art they are con-

tent, if only truth develops. Knowledge can wait, but art is impatient. Art represents something to be done. It is wholly dependent upon knowledge and intelligence, and its demands upon science are constant. Art is always seeking more knowledge, and its appetite can never be satisfied.

There are many kinds of art, and the healing art differs from all others, the fundamental difference being philosophical. The philosophy of technology and its branch, prosthesis, has no essential relationship to the biological art of healing, notwithstanding that prosthesis may be employed contemporaneously with therapeutic efforts. And, while dentists have learned much of medical *science* and can discuss many subjects which are exclusively within the field of medicine—(not medicine the art, however, but medicine the science)—few have comprehended the *principle* of medicine, "the healing art." Art, when comprehended as something to be done, is related to the idea of "work." All art requires material and it is the character of the material which identifies the nature of each particular art. The technologist knows from experience that his material is inert, and that his own life force must be contributed to that material in order to proceed with the "work." The established premise of that philosophy is *material cannot work.* The philosophy of the healing art denies the premise of technological philosophy and asserts *material can work.* These differences are basic.

When the healing art was very young, medicine adopted a Latin formula, *vis medicatrix naturæ.* From that principle developed the idea which gave recognition to "the capability of living tissue, animal or vegetable, to remedy or remove disease, or to repair injuries inflicted upon it." That idea identifies the principle of "healing." To aid nature in accomplishing that purpose identifies the healing art. Health is a term which identifies the objective of the healing art. It is life present in the material of the healing art—an organism, which identifies the force, and the force is life, vital force, resident in the material. But the idea is not complete without giving recognition to *vis preservatrix*—a principle which maintains health before disease attacks the organism.

The transfer of interest from the inert material of prosthesis, to a cadaver, was an easy ideological step. The philosophy of technology is applicable to anatomical study, for a cadaver may be "cut apart" and objectively studied from the perspective of technology and no principle of that art is violated. Likewise, when the dentist takes an impression of the teeth or of the edentulous jaw, and "pours" a cast, he tends to confuse the inert cast with the vital tissue from which it was derived. He then may make the serious error of thinking that any appliance which he may fit to the cast will be acceptable to the living tissues of the mouth. That the prosthesis so constructed is often tolerated by those living tissues is striking evidence of the marvelous forbearance of Mother Nature. It is, however, an easy step from the study of anatomy to a study of the injuries that come from disease, as found in a cadaver. But the idea that these scars (which are the results of an incoördination of physiological action) are *not* disease, is a very difficult idea for a dental student to accept.

But morphology of the body or its parts was comprehensible and that perspective was transferred as an acceptable idea to dental pathology. But it is only admissible to the subject of disease while life exists in material. And an organism can no more be dissected than a cadaver can receive surgical treatment and recover life, or than health can be developed by fabrication.

That observation will serve to introduce a discussion of dental surgery. To avoid confusion over the use of terms, it should be recognized that "oral surgery" validly *is* surgery; also that much "dental surgery" is not surgery, but a traditional development from a technological philosophy. When the principle of surgery is applied to a living body, recognition is given to vitality and that idea has priority over the principle of surgery. Omit the idea of "the healing reaction to injury" and surgery does not exist. "Good" cannot proceed from "bad." Only when the means is good, in ethics or morals, can we accept the idea "the end justifies the means"—a syllogism cannot begin with a conclusion—no more than can physical life begin in death.

Periodontia is an interest of the healing art. At present the greater thought is being given to periodontal disease. But periodon-

tal health is actually the greater interest; especially it is of a greater interest to the patient. But health cannot be mistaken for an object, notwithstanding the idea that both disease and health can be comprehended as physical condition. When disease is mistaken for an object, and it usually is, very unsound deductions are made and very misleading ideas intrude into the scheme called "treatment."

Pyorrhea can be "eradicated" under the principle of dental surgery. A so-called "surgical method" can be popularized. The amputation of the gingiva is possible, but as a "cure" it is not nearly as successful as extraction of the tooth. But that scheme of "treatment of disease" has no relation to the principles of the healing art. "Surgical interference," as the phrase implies, is physical insult. Most interference is insult. The executioner's axe is not surgery, even though decapitation is an operation involving the cutting of vascular tissues. Nor can periodontists be classified as "radical" operators in that sort of surgery and contrasted with those who condemn their ideas, whom they call "conservative operators." This so-called "radical method" is indeed "eradication" of diseased tissue which has been mistaken for the disease itself. Were the "witches" who were burned at the stake guilty? Or, were the conservative and religious Puritans radicals? Were the Puritans not mistaken in their philosophy? This writer thinks kindly of all deluded people and he is not too sure of himself. But these ideas are basic principles of medicine, not the result of mental gymnastics developed in solitude. You are challenged to prove them wrong—then we'll all learn something.

The healing art as represented by medicine had had a struggle for centuries to produce the knowledge which provided a background for medicine. The earlier physicians did not take kindly to surgery in its early days, but the "physician and surgeon" did finally establish himself in medicine. Looking backward a century, is it not obvious why medicine failed to establish a union with dentistry? It is more difficult to unteach accepted principles than to instruct a neophyte. Dentists did not know that they possessed a technological philosophy; some do not know it yet. Some day they will know. But therein lies the reason for past and present day confusion.

Dentistry will some time clearly become a "branch of the healing art, coequal to a specialty of medicine." And periodontists should lead the way. For the periodontist, more truly than even the oral surgeon, practices the healing art. Because of this fact, the periodontist, if he works with nature, induces healing of diseased tissue—while the oral surgeon must remove the diseased parts in order to quickly get rid of the disease. But both the periodontist and the oral surgeon "work" under the philosophy of *vis medicatrix naturae.* On the other hand, the dentist who fills a cavity or constructs a bridge or a denture, necessarily works under the philosophy of technology. This is no disparagement of that procedure. Once the cavity has developed there is no other known way of "saving the tooth." The unfortunate thing is that filling cavities does not lead to any constructive thought as to their initial development. And it is for that reason that dentists so consistently think of prevention of decay in terms of filling small cavities to prevent their becoming big ones. We have not yet reached the idea of prophylaxis.

Periodontia must bridge this gap between technological dentistry and biological dentistry, and then it must bridge the gap between all dentistry and medicine. It is here that I see the future of periodontia. We have learned "how" in the past twenty-five years to bring health to diseased mouths, now we must learn "why." I now see periodontology going on to trace back to their inception the factors which we know are responsible for periodontal disease. The prevention of periodontal disease in years to come will not belong to the periodontist, it will belong to the dentist of the future, who will so supervise the general health and the oral health of his patients from earliest childhood on, that periodontal disease and dental caries, too, will never occur. That is an ideal. And, leading up to that day, we shall see the periodontist moving closer and closer in his philosophy and in very fact, to the physician. And signs are not lacking that as he moves in that direction; the physician will not only lend him a helping hand when he matures, but will welcome him into the world's noblest fraternity, that of *the healing art.*

[*This symposium on the Historical Background of Periodontology will be continued in July, 1939 issue of The Journal of Periodontology.*—Ed.]

The Responsibility Is Ours

IT HAS recently been called to our attention, that the list of regular subscribers to the only dental journal in this country devoted exclusively to the reports and records of scientific research in dentistry is unbelievably small. So small in fact that we lack the courage to publish the number, yet at the same time so significant, that an inquiry into the cause of the apparent disinterest in a project of such consequence to dentistry, seems not only justifiable but obligatory.

Dental patients require, even demand, that we relieve pain associated with their teeth and perform all operations on and about the teeth not only adequately but with skill. The "sledge-hammer dentist" so-called, is out so far as the public is concerned. The successful practice of dentistry is not so dependent upon our knowledge of the last word in scientific research as it is upon the individual operator's judgment in diagnosis and his ability to apply the correct principles of operative dentistry in the oral cavity with accuracy and skill, and with humanitarian consideration for the patient. We may or may not have accepted the latest theories regarding dental caries, erosion, or periodontal diseases, but the chief concern of our patients is whether we can keep them dentally comfortable, preserve and skillfully restore their teeth, and replace them satisfactorily if lost. It is quite natural then that dentists should be primarily interested in the type of dental literature that will improve their services for patients. Unfortunately, many of us have acquired the habit of looking for these grains of wisdom in the "throw-away" dental publications, for they are such easy reading and cost us nothing.

It has been said many times that "the best things in life are free," such as air, sunshine, and water. The handicap in this is that we do not really appreciate things received without some personal effort. We are apt to forget that the subscription price for many of our dental journals is included in our dues to the respective dental societies publishing them; consequently, when someone suggests that we pay a direct subscription price for a dental journal, we demand to know what we are going to get out of it.

We have been led to believe in recent years that we are members of a scientific profession and our heads have gone up and we have been filled with pride. Most of the scientific research in dental fields has cost us little if anything; yet we and our patients have been the most benefitted by it. It has been carried on by the personal sacrifices of the very few, only occasionally financed by philanthropy and but meagerly by educational institutions. Its value to methods of dental practice generally can be readily imagined by recalling the contributions of Drs. G. V. Black, W. D. Miller, William Taggart, and others as well known.

Two years ago we were asked, as members of a scientific profession, to aid in continuing the Journal of Dental Research, which had been recording the

researches related to dental problems and which had been edited and published at no cost to us but largely through the sacrificial efforts of one man—not a dentist, but one deeply interested in dentistry. To what extent we have shouldered this responsibility is shown by the small subscription list which, in proportion to the membership of the American Dental Association, is something of which we cannot be proud. It definitely reflects upon the dental profession as a whole. In the minds of the lay person or the medical profession, respect for dentistry as a scientific profession would not be much enhanced if the above facts were known to them. We have not been asked to contribute to the research but merely to aid in sharing the expense of recording the findings, surely a small request, by subscribing to the Journal of Dental Research.

It is not a question of whether or not we have the time or desire to read the Journal. That is beside the point. Let us swell the subscription list with regular, sustaining and life subscriptions until we feel that we are assuming our rightful privilege and responsibility. In this way we shall be stimulating those researchers who have been willing to carry on with little encouragement and who have been so greatly handicapped without our coöperation. *The responsibility is ours.*

—G. R. S.

References

THE VALUE of accurate and complete references in our current dental literature has been appreciated only in recent years. When these references are accurate and complete, they add immeasurably to the reliability of the article itself, as well as to the reader's possible sources of information on the subject under discussion.

It is imperative, however, that *complete* references be furnished by the author, since the editor often does not have adequate library facilities available and, being otherwise handicapped by lack of time and secretarial assistance, he may not be able to locate the author's references. Inaccurate references are annoying to the reader and to librarians as well as to the editor.

If a book citation is used, it is essential that the author's full name be given and the exact title of the book, as well as other information, especially the year of publication and the pages of reference. In periodical citations the volume number, page, month, and year are all important as well as the author's name. Inaccuracies are bound to creep in, especially if original copy was not clear or on account of errors of the typist or careless proofreading.

The reader is referred to pages 56-58 of this Journal for helpful information on the subject of references and abbreviations and he is urged to coöperate with the effort of the American Association of Dental Editors to establish uniformity in references and abbreviations in dental literature.

—G. R. S.

Traumatic Occlusion and Gum Inflammation*

By BALINT ORBAN, M. D., D. D. S.,† *Chicago, Illinois*

THE question of the importance of traumatic occlusion in producing pathologic conditions in the supporting structures of the teeth has been frequently discussed in dental literature in recent years. We must differentiate between three types of papers on the subject. One type was concerned purely with the histopathologic study; the second, with only the clinical phase of it; the third tried to correlate the histopathology with the clinical picture. All these studies, whether correct or not, were contributions to the development of our present-day knowledge.

It is quite commonly accepted today that the supporting structures of the teeth are in close functional relationship to each other. The cementum covering the surface of the root, the peridental membrane, and the alveolar bone can be considered as a functional unit. All functional changes in the dentition will be transmitted to the supporting apparatus, and all these changes will have their influence on the structure of these tissues. The entire bony tissue of the skeleton is a functional structure and so is the bone of the jaws. Functional changes in the dentition will have their influence on the entire architecture of the jaws. This holds true, of course, not only for cases of over-stress, but also of reduced stress, or so-called disuse. I do not want to leave the impression, however, that I consider functional changes the only cause for disturbances in the normal relationship of the supporting mechanism of the teeth. The teeth are an organic part of the entire body, connected with the body by connective tissue, epithelium, blood vessels, lymphatics, and nerve fibers; and all diseases of the body may have their influence on the structure of the teeth, especially on the supporting mechanism. Not only diseases, but hereditary factors, general metabolic conditions, and what we, in one word, express as constitutional factors, will have their influence on the teeth and their relationship to the supporting structures. The function of the ductless glands has a definite influence on bone formation, calcification, eruption of teeth, condition of mucous membrane, etc. We should never consider the teeth as single structures, exposed only to local influences, but we must regard them as a part of the entire body with all the advantages and disadvantages of this relationship.

In coöperation with Gottlieb[1] we conducted experiments over a period of five years to determine the pathologic changes which take place under the influence of undue stress and also disuse. Some of the results of this investigation have been critically reviewed by Charles F. Bödecker[2] and also taken into consideration in a book by R. Kronfeld[3]. A short report has also been published by Gottlieb and Orban in the *Journal of Dental Research*.[4] Nevertheless, I feel the necessity of giving a few of the results of these experiments at this time.

Fig. 1—A set of dog's teeth. The bite is raised by placing crowns on the first lower molar and the fourth premolar in the upper jaw. By this arrangement the upper tooth was pressed to the buccal, while the lower tooth was pressed against the lingual side.

In Fig. 1, a set of dog's teeth is shown at the beginning of an experiment. High cast crowns have been placed on the large first molar of the lower jaw and fourth premolar in the upper jaw on the right side. The left side did not carry any crowns. The crowns had inclined planes so that the upper teeth were pushed toward the buccal side while the lower teeth were pushed toward the lingual side. As shown in the pic-

*Read at the Twenty-Fifth Annual meeting of the American Academy of Periodontology, October 21, 1938, St. Louis, Missouri.

†Northwestern University Dental School.

ture, the bite was raised so that the jaws could not be closed at all and the entire load of the bite rested on the two teeth with the crowns. The tissue changes in the supporting structures were studied from twelve hours' duration to thirteen months'. By this plan we could follow up the tissue changes which occurred, due to the trauma, from the first day to several months. Young, middle-aged, and old dogs were used to gain information regarding the difference in tissue changes at different ages. We had dogs under experiment for 12 hours, 1 day, 36 hours, 2 days, 60 hours, 3 days, 4 days, 6 days, 1 week, 10 days, 2 weeks, 15 days, 20 days, 3 weeks, 4 weeks, 1 month, 1 month and 6 days, 1 month and 15 days, 1 month and 18 days, 2 months and 10 days, 2 months and 24 days, 3 months, 3 months and 5 days, 3 months and 7 days, 3 months and 12 days, 4 months, 4 months and 9 days, 5 months and 10 days, 6 months and 12 days, 11 months and 18 days, and 13 months. Some of the short duration experiments (12 hours to 3 days) were carried out on the front teeth by orthodontic measures —bands, arch wire, and ligatures.

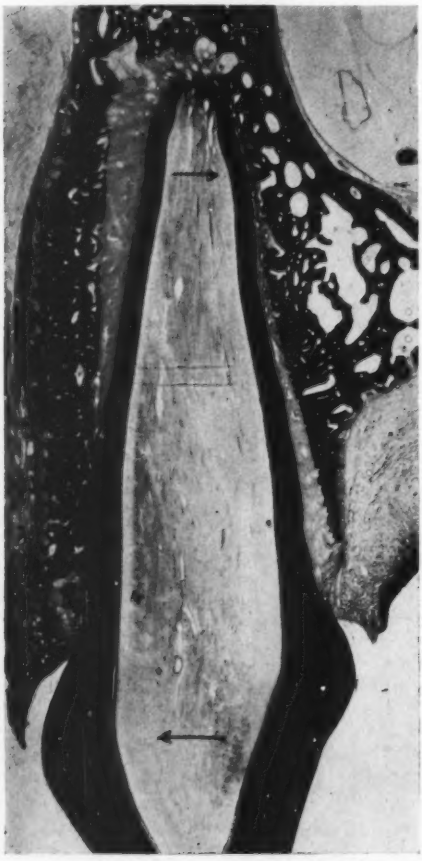


Fig. 2—Position of the distal root of the upper premolar in the alveolus after carrying the crown for 36 hours. The peridental membrane is compressed at the alveolar margin on the labial side and at the apex on the lingual side. On the opposite sides the peridental membrane is considerably wider than normal. The arrows indicate tooth movement.

In addition to these dogs (there were usually two for each period), we had the opportunity to study the tissue changes of trauma in monkeys which were used by C. Breitner[5] for orthodontic experiments. Eight monkeys were used in this study. Disuse was studied in rats by O. Preissecker.[6]

At the time the above experiments were finished we had only a few human jaws showing traumatic changes[7,8] similar to those found in dogs, monkeys and rats. Since that time we have studied a large number of human jaws to investigate traumatic changes and found the same disturbances in a very large number of cases.[9]

The immediate result of placing the high crowns on the teeth was a tipping of the lower teeth toward the lingual, and the upper toward the buccal.

In Fig. 2 we can see the root of an upper tooth which carried the crown for 36 hours. The peridental membrane is compressed at the alveolar margin on the labial side and at the apex on the lingual side, while on the opposite sides the peridental membrane is considerably widened. The axis around which the tooth has tipped is slightly apical from the middle of the root. This is true whenever a tooth is tipped. The tipping axis is never at the apex of the tooth even when the slightest forces are used.[10]

Traumatic changes could also be observed in the peridental membrane of the same teeth on the left side of the jaw. They were caused by cusp interference of these teeth, due to raising the bite on the right side and pushing the lower jaw to the left side. These traumatic changes were present in practically all cases on the left side of the jaw but were less extensive than on the right side.

In Fig. 3 we see an upper incisor of a young dog which was ligated to an orthodontic arch wire for 48 hours. The tipping of the tooth is quite evident from the narrowing and widening of the peridental space. The tissue changes which have been

Fig. 3—Upper incisor of a young dog. This tooth was ligated to an orthodontic labial arch wire for 48 hours. Note the compression of the peridental membrane at the alveolar margin on the labial side and at the apex on the lingual side. The opposite peridental membrane areas are widened. The arrow indicates the direction of force.

introduced by this trauma can be observed in higher power pictures.

Fig. 4 shows the alveolar border of the labial side. This is the pressure side. The peridental membrane at the alveolar margin has been completely necrosed by this trauma. It has lost its tissue-staining reaction and is black without any cellular structure. Of course, this necrotic tissue can not stay indefinitely there and must be removed. Also, the bony alveolar crest

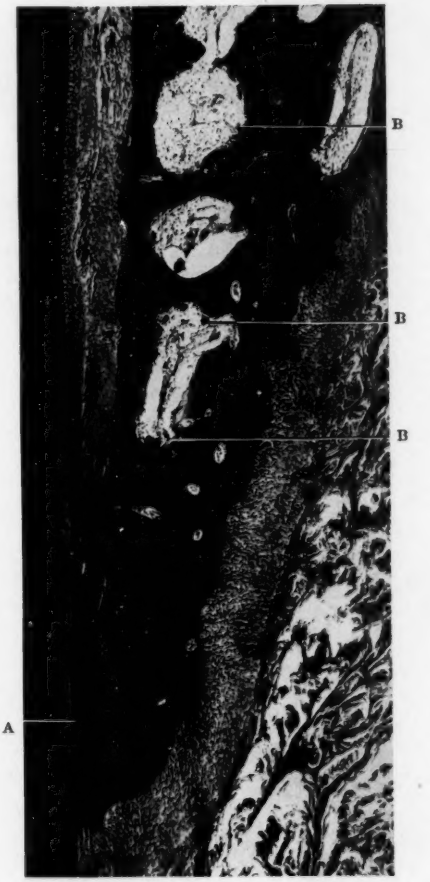


Fig. 4—Higher magnification of the labial alveolar margin of the tooth seen in Fig. 3. The peridental membrane is completely compressed and necrotic (a). In the adjacent marrow spaces giant cells have developed and resorption of the bone has started (b).

must be removed by resorption. This resorbing process starts near the necrosis area but where the pressure has not been definitely severe. In the picture under discussion the resorption is already started in the marrow spaces close to the necrotic area. In the experiments to be discussed here we shall see that the resorption process will remove the entire necrotic area and bony crest standing in the way of the moving tooth.

The labial side of the peridental membrane at the apex is the tension side (Fig.

5). We can observe the widening of the peridental space but no tears are to be seen. The most remarkable feature of this specimen is the extensive bone-forming process taking place only 48 hours after the start of the experiment. Two days after the tooth had been ligated to the arch wire, we found all along the side of the alveolar bone fine spicules of new bone projecting toward the tooth and surrounded by masses of osteoblasts. We can definitely picture the outline of the alveolar bone as it would have been if the tooth had been kept in this position a few days longer.

Fig. 5—Higher magnification of the tension side at the apex (a) of the tooth shown in Fig. 3. The peridental membrane is considerably widened. Along the dense connective tissue bundles, where a large number of osteoblasts have developed (b), bone formation has started.

In 60 hours (Fig. 6) the resorption process is largely increased. Numerous giant cells are at work to remove the bone in the way of the moving tooth. The necrotic tissue is also undermined by the resorption process which, in this stage of trauma, is in progress in the marrow spaces as well as in the peridental membrane near the necrotic area. Of course it is quite evident that the necrotic peridental membrane cannot produce any giant cells, or other cells, because necrotic tissue is not capable of any activity. For this reason no bone resorption can take place adjacent to the necrotic area. The larger the necrotic area, the longer it may take to remove the bone and necrosed tissue.

If the necrosed area is relatively small the resorption process may be finished in 10 to 16 days, as seen in Fig. 7. The bite-

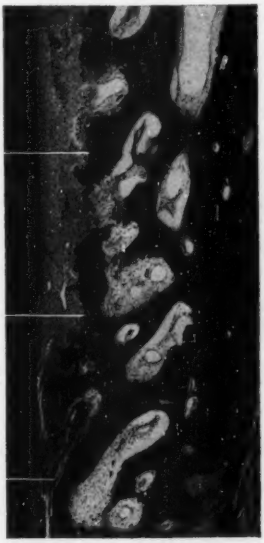


Fig. 6—Dog's tooth in trauma for 60 hours. There is compression and necrosis of the peridental membrane at the alveolar margin (a). A large number of osteoclasts (giant cells) are occupied in absorbing the bone (b) to relieve pressure.

raising crown was in action in this case for 14 days and we can observe that only a very small necrotic tissue area is present; and the bony socket, which prevented the tooth from further movement, is almost entirely eliminated. Only a group of giant cells indicates the site of former traumatic injury.

If the traumatized peridental membrane is of some length, the cleaning-up process may take a longer time, as seen in Fig. 8. In this case the cap was on for 15 days but still large masses of necrosed tissue are left behind. Large amounts of bone must also be removed before the hindrance to further tooth movement is eliminated. In this specimen, for the first time, we observe bone formation outside the alveolar crest, as a measure taken by nature, to prevent shortening of the alveolar ridge. New bone is formed on the surface of the alveolar bone —fine trabeculae with small fibrous marrow spaces. The new formation of bone is more

rapid at the crest. Here it is the widest and becomes gradually narrower toward the apex. This seems to be due to the intensity of pressure in the peridental membrane.

The marrow spaces in the old alveolar crest are wide while the bone toward the peridental membrane is almost all resorbed. Only a thin layer of bone is seen limiting the marrow spaces toward the compressed peridental membrane. At the apical end of the picture the peridental membrane is wide because at this point the pressure was not so strong as to necrose the tissues and resorption took place, not only from the marrow spaces, but also from the peridental membrane.

Fig. 7—Dog's tooth in trauma for 14 days. The traumatized peridental membrane and bone have been largely removed by the activity of the giant cells. Necrotic tissue and giant cells can be observed only at one small area (a).

Fig. 9 is a good example of the resorption and new formation process. It shows the apex of a tooth which was over-loaded for 3 weeks. The resorption process is almost finished; only a few osteoclasts indicate the former necrotic area. At the other side of the root new formation of bone took place, probably to the same extent as the tooth had been moved.

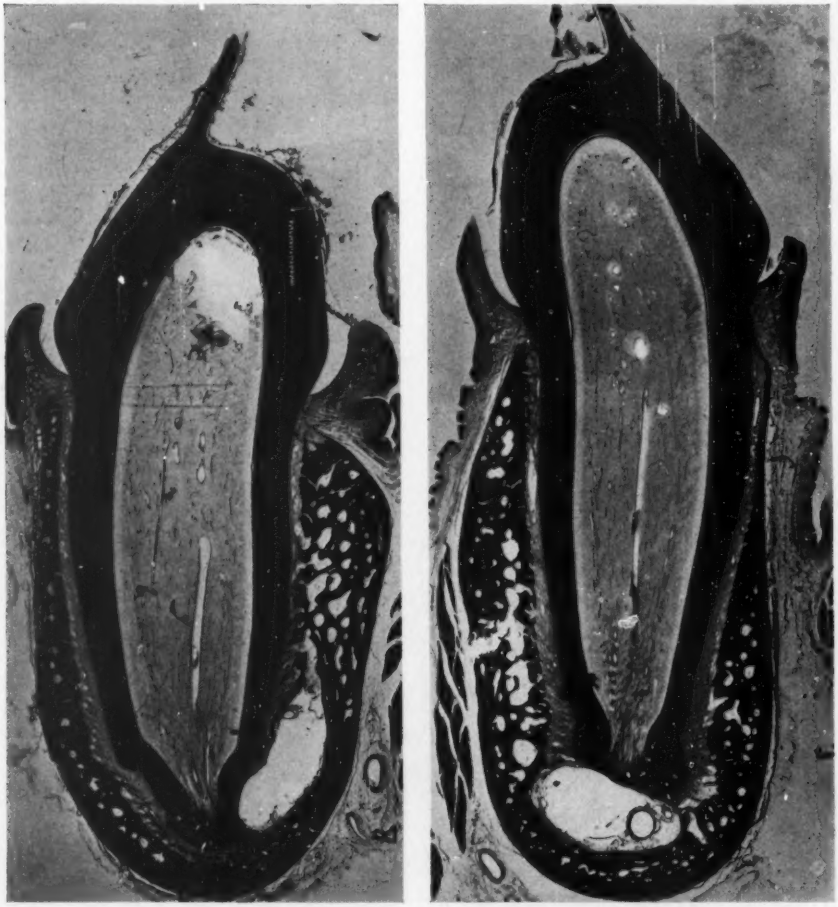
Fig. 8—Dog's tooth in trauma for 15 days. The peridental membrane was compressed and damaged over a long surface and it therefore takes more time for the giant cells to remove all the compressed tissues (a). At the outside of the alveolar margin bone formation took place (b), compensating for the resorption of bone from the inside.

The overload can be very heavy and it may move a tooth some distance; but if the pressure is relieved, either by removing the appliance or by shifting the tooth out of traumatic reach, the tissues soon recover

Fig. 9—Apex of a dog's tooth in trauma for 3 weeks. The necrotic pressure area is almost eliminated. Only a small group of giant cells indicate the former region of high pressure (a). On the tension side, a new layer of bone (b) indicates the amount of tooth movement during the three weeks.

and are restored to normal functioning structures. I will show one case which will exemplify the above statement.

In Fig. 10 we see the right (10a) and left (10b) tooth of a dog which was in experiment for 2 months and 10 days. The end-result of this experiment is shown in the picture. There is considerable shortening and widening of the alveolar bone on the right side and compression of the apex of the same tooth. The apex is compressed because the experiment started when the apex was still open—the root not being fully formed. During the experiment the root formation progressed and, because of the traumatic pressure, was crooked when formed. In spite of the fact that the alveolus on the right side is considerably shorter than that on the left, we cannot see any traumatic changes in the peridental membrane. According to our experimental experience, it is quite evident that the same changes had to take place as have been shown in the foregoing pictures. Not once,



A B

Fig. 10—a and b. Roots of the right (a) and left (b) molar teeth of the same dog. The apex of the crowned tooth (10a) is crooked because it was undeveloped at the time of the experiment. There are no other signs of trauma in the peridental membrane. The gum margin is normal. The time of trauma was 2 months and 10 days. Note the difference in length and width of the alveolar bone and the location of the mandibular canal on the right and left sides.

but several times, were these changes repeated with the end-result shown in Fig. 10a. The tooth moved out of occlusion—out of trauma—and at the time the dog was sacrificed, there was no trauma and the entire alveolar bone and peridental membrane had regained their normal functional appearance. Also, the epithelial attachment, gingival crevice, and subepithelial connective tissue are in normal structural condition. No signs of pathology are to be noted in this region.

These few pictures tell the story of our traumatic occlusion experiments. Of course, there were many individual variations in the dogs. Not all reacted in exactly the same way; but the result has been shown in these few cases. In older dogs we sometimes found not only resorption of the bone but also resorption of the cementum. However, the cementum resorption is not nearly as extensive as the resorption of the bone. Several times, in the older dogs we found ankylosis of the teeth with the alveolar bone after resorption of the cementum occurred. In most of our cases the gingival margin of the overloaded teeth was normal. Only a few times, and in older dogs and long experiments, did we find some gum disturbances, such as inflammation of the free margin of the gum and pocket formation. This pathology always occurred on the side from which the tooth was moved—that is, on the side of tension. The explanation for this fact was very simple, the cause being food impaction. The two crowns pushed each other in opposite directions, the lower being pushed toward the buccal side and the upper toward the lingual side. The occlusal edge of the crown of the upper tooth wedged the food into the pocket on the lingual side of the lower tooth, especially after the tooth was tipped extensively toward the buccal side. We have seldom seen any gum disturbance in the upper teeth as they are tipped less than the lower, having three roots while the lowers have two. We also observed less food impaction in upper teeth, as the occlusal edge of the lower crown was tipped buccally and the food could not be pushed into the buccal gingival pocket of the upper tooth.

In some cases irritation of the gum margin occurred as food debris would catch on the margin of the cast crown. No signs could be detected to indicate that the traumatic injuries in the peridental membrane would bring about any changes in the gum margin or cause pocket formation. On the pressure side, we would have expected changes due to compression of the peridental membrane, and therefore disturbance in the blood and lymph circulation; but the histologic picture did not reveal any changes. On the tension side, only in extremely dislocated teeth, could pocket formation be found and, as stated before, this pocket formation was not caused by traumatic changes in the peridental membrane, such as tears of the fibers, or similar conditions, but could be traced to food impaction.

These pathologic facts seem to be in direct opposition to clinical observation by many of the leading periodontists who claim to have found a definite correlation between trauma and pocket formation. I spent considerable time at the microscope, as well as in examining patients, to find the explanation for the difference in observation and opinion and I came to the following conclusion, which I hope will satisfy the clinicians as well as the research men.

Traumatic occlusion and gum inflammation, from plain gingivitis to deep pocket formation and suppuration, are two pathologic conditions which are very frequently found in human beings. It may be safe to state that about 90% of all people, at least in the civilized world, have signs of both of these disturbances. There are a few individuals who have a completely ideal occlusion and articulation, but most people have some divergence from the normal, such as missing teeth, changed or false mesio-distal or bucco-lingual relationship, extensive overbite, fillings, crowns, bridges—fixed or removable—and so on. These are the conditions that bring about traumatic occlusion. If we consider, in addition to these more or less outside influences, the biologic factors, such as continuous active eruption of the teeth, the wearing off of the contact points, mesial drifting of the teeth, difference in hardness of the teeth, attrition, and other disturbances, then it is quite clear that traumatic occlusion is one of the most common disturbances of the denture. The same is true of the gum disturbances. Calculus formation, improper brushing technique, injury from food, the tooth brush or some other instrument, irritation by restorative dental work, mouth breathing, etc., are the most common extrinsic factors causing trouble with the gums.

Neither should we forget the biologic factors, such as the continuous passive eruption of the teeth, and thus recession of the gums, which in one case may be slow and in another fast, regular, or irregular. We also know that many of the general metabolic disturbances and changes in the inner secretory gland correlation have their influence on the mucous membranes. Poisonous substances are often desposited in the gums, and general catarrhs of the mucous membranes also affect the oral cavity. There are doubtless several other important etiologic factors in traumatic occlusion, as well as in gum disturbances. This, however, does not change the fact that both of these conditions are very frequent, and, as stated previously, affect a large percentage of individuals.

Is it any wonder, then, that both traumatic occlusion and gum disturbances are frequently found in the same mouth? The reverse is the exception. I believe this is the reason that both conditions have been found in combination and it *seems* reasonable to believe and state that one is a result of the other. I am sure it will always be difficult for a clinician to separate the two conditions clearly as the field on which both act is small and, topographically as well as functionally, they are very closely related. However, if we combine our clinical experience with our research findings, we can clearly and definitely separate these conditions—traumatic changes and gum disturbances. They each present such different pathological pictures that their separation is not difficult. I have shown the traumatic changes but in this presentation I have omitted a discussion of gum conditions as I should like to take that subject up in a special treatise. Gum conditions present themselves in different forms and stages of inflammatory reactions and their consequences.

In my attempt to separate traumatic occlusion from "pyorrhea," may I use a comparison which might help in understanding our problem. A tuberculous individual may become infected with another infectious disease; for example, typhoid fever. These diseases are definitely different and basically have nothing to do with each other; but it is clear that if both attack at the same time they are much more difficult to handle. One disease may be combated with good results but if both are present the patient's chances for recovery are slight. The patient should be treated according to the most urgent needs and both diseases must be cared for. It may be necessary to treat one disease first while the other must temporarily be neglected. The same is true of traumatic occlusion and "pyorrhea." A tooth in trauma and with inflamed gums is like the individual with tuberculosis and typhoid fever. While they are two different diseases with regard to their nature, they spoil conditions for each other. Trauma will cause bone resorption due to pressure, and inflammation will also cause bone resorption due to circulatory disturbances and toxic influences. Both must be treated if both are present; in treating only one we may fail.

Inflamed tissue needs rest to heal properly, and traumatized tissue needs a good blood supply and a healthy environment to be repaired. Clinically, it may be difficult to determine which was first—the trauma or the inflammation—as they may both be active at the same time; but one can precede the other and each may be a predisposing factor for the other. A traumatized tissue may more easily become infected and inflamed and an inflamed tissue will yield much more easily to trauma. I may even state that every tooth which has lost some of its supporting structures because of inflammation is in trauma, even if in normal occlusion, because the loss of some of the bone and peridental membrane loosens the tooth and weakens the structures in resisting the load. The same is true of teeth which become loose from trauma. It is a common observation that tartar will deposit much faster around loose teeth than around firm teeth in the same mouth. This is due to the fact that the loose teeth are avoided in chewing as well as in brushing; and these resultant unhygienic conditions will much more easily produce a pathologic reaction in the soft tissues around the loose teeth than around the firm teeth, as the gum is traumatized not only by the tartar but also by the back and forth movement of the tooth. The attachment may be severed, and in that way the pocket deepened, which again gives more opportunity for infection and gum irritation. The two conditions are combined in a vicious circle, promoting injury and preventing healing.

Conclusions:

Traumatic occlusion produces injuries in

the peridental membrane with subsequent resorption of the alveolar bone and widening of the peridental membrane. Thus, traumatic occlusion produces pathology in the peridental membrane.

"Pyorrhea" is an inflammatory reaction of the gum margin, largely due to local irritation. This irritation, combined with the inflammation, will produce deepening of the gingival crevice and induce bone resorption.

Traumatic occlusion and local irritation are very frequent causes of development of pathologic conditions—possibly in 90 per cent of all people. That is why we usually find traumatic changes and inflammatory reactions at the same place and at the same time.

REFERENCES

1. Gottlieb, B. and Orban, B. *Tissue Changes by Experimental Overstress.* G. Thieme, Leipzig. 1931.
2. Bödecker, Ch. F. Critical Review of Gottlieb and Orban's *"Die Veränderungen der Gewebe bei Übermässiger Beanspruchung der Zähne,"* Leipzig. 1931. *Int. J. Orthod. Oral Surg. & Radiogr.* 18(9): 895. Sept., 1932.
3. Kronfeld, R. *Histopathology of the Teeth and Their Supporting Structures.* Lea & Febiger, Philadelphia. 1933.
4. Gottlieb, B. and Orban, B. Tissue Changes in Experimental Traumatic Occlusion with Special Reference to Age and Constitution. *J. D. Res.* 11: 505. 1931.
5. Breitner, C. Tissue Changes Caused by So-called Bite-raising Plate Acting on the Front Teeth. *Zeitschr. f. Stomatologie* 30: 1185. 1932.
6. Preissecker, O. Influencing the Peridental Membrane by Experimental Disuse. *Zeitschr. f. Stomatologie,* 29: 442. 1931.
7. Orban, B. Tissue Changes in Traumatic Occlusion, *J. A. D. A.,* 15(11): 2090. Nov., 1928.
8. Grohs, R. Changes in the Human Peridental Membrane Due to Overstress. *Zeitschr f. Stomatologie* 29:386. 1931.
9. Orban, B. and Weinmann, J. Signs of Traumatic Occlusion in Average Human Jaws. *J. D. Res.* 13: 216. 1933.
10. Schwarz, A. M. Movement of Teeth Under Stress. *Zeitschr. f. Stomatologie,* 26:40. 1928.

180 N. MICHIGAN AVENUE

Twenty-Sixth Annual Meeting

AMERICAN ACADEMY OF PERIODONTOLOGY
MILWAUKEE, WISCONSIN
JULY 13-14-15, 1939

THURSDAY

Morning

REPORTS OF OFFICERS AND COMMITTEES:
Secretary-Treasurer. Raymond E. Johnson.
Committee on program. Samuel R. Parks.
Committee on clinics. Robert L. Dement.
Committee on general arrangements. Lynn A. Fonner.
Committee on scientific investigation. Dickson G. Bell.
Committee on nomenclature. Arthur H. Merritt.
Committee on publication. Harold J. Leonard.
Special committee reports. W. A. Beatty, Harold J. Leonard, Carlos H. Schott, Samuel R. Parks, Juanita Wade, Justin D. Towner, Russell A. Sand, Harry Lyons, Willa Yeretsky, Grace Rogers Spalding, M. Monte Bettman and Edward B. Spalding.
Presidential address. A. W. Bryan.

Afternoon

Forty years of experience in periodontia. James O. Hall, Waco, Texas.
Periodontal disease in children. Konrad Lux, Waco, Texas.
Habit—an etiologic factor of periodontal disease. Sidney Sorrin, New York City.
Vincent's infection: retrospect, suspect and prospect. Fred D. Francis, Iowa City, Iowa.
Periodontal sepsis in relationship to systemic

disease. Russell A. Sand, Fargo, North Dakota.

FRIDAY

Morning

Past-presidents' breakfast.

Report of past-presidents' meeting. Austin F. James, Chairman.

Committee on scientific investigation. Dickson G. Bell, Chairman.

Periodontic problems in the child patient. John C. Brauer, Iowa City, Iowa.

The effect of magnesium deficient diets on oral and dental structures. Hermann Becks, San Francisco, California.

Report of the sub-committee on scientific investigation. Clayton H. Gracey, Birmingham, Michigan.

Afternoon

ROUND TABLE DISCUSSION LUNCHEON

Leaders: Russell A. Sand and Harry Lyons.

1. How could one justify the thought that bone calcium applied locally could assist in building new cementum and bone? Austin F. James, Chicago, Illinois.
2. Has oxygen insufflation a place in periodontal treatment? M. Harry Garvin, Winnipeg, Manitoba.
3. The advantages of reconstruction following treatment of periodontal lesions in dental cripples. C. W. Hoffer, Nashville, Tennessee.
4. Are there any physiologic means of keeping the mouth and teeth clean and sweet besides mechanical brushing? Harold J. Leonard, New York City.
5. What is the status of periodontia in dental practice today? Clyde C. Sherwood, Toledo, Ohio.
6. If partial dentures hasten the loss of remaining natural teeth, what is the reason? Edward B. Spalding, Birmingham, Michigan.
7. How can the general practitioner best be taught to recognize and treat periodontal disease? M. Monte Bettman, Portland, Oregon.
8. Which type of periodontoclasia may be considered as a definite focus of systemic disease? Walter H. Scherer, Houston, Texas.
9. Can we distinguish between periodontoclasia of systemic or local origin? Carlos H. Schott, Cincinnati, Ohio.
10. Why do we ladies like to attend the Academy meetings each year? Mrs. Harold J. Leonard, New York City.

CLINICS

Robert L. Dement, Chairman

Fundamentals of physiology and pathology of the periodontal tissues, with special reference to prophylaxis and therapy of periodontal diseases. Rudolf Kronfeld, Chicago, Illinois.

Technique of sub-gingival curettage with pre- and post-operative radiographs. Walter Leabo, Shreveport, Louisiana.

For better function and esthetics improve the symmetrical end arrangement of the anteriors. Luther A. Neil, El Paso, Texas.

A consideration of the possible beneficial effects of precision type bridges on the periodontal structures. R. L. Wescott, Evanston, Illinois.

Equilibration of occlusion. Sidney Sorrin, New York City.

Evening

ANNUAL BANQUET

SATURDAY

Morning

Possibile occlusal factors in relation to periodontal structure. Allan G. Brodie, Chicago, Illinois.

INCIDENTS OF OFFICE PRACTICE

1. A case of periodontoclasia before and after treatment. W. F. Andes, Miami, Florida.
2. A case of paradentosis complicated with Vincent's infection. Marion H. Gray, Little Rock, Arkansas.
3. Inflammatory reaction following treatment of cancer of the lip. R. Dean Creamer, Phoenix, Arizona.
4. Oral manifestations of monilia. Walter H. Scherer, Houston, Texas.
5. A case report. T. E. Braly, Chattanooga, Tennessee.
6. Excessive gingival hypertrophy reduced by occlusal adjustment. William Y. Hayden, London, Ontario.
7. A case report. G. P. Robertson, San Antonio, Texas.
8. Treatment procedure of the complex pocket. Oscar Bjorndahl, Saint Paul, Minnesota.
9. Periodontal progress. Edward L. Ball, Cincinnati, Ohio.
10. The value of clinical observation. Hunter S. Allen, Birmingham, Alabama.

An Experimental Study of the Use of Sodium Sulphid Solution in Treatment of Periodontal Pockets*†

By FRANK E. BEUBE, D. D. S., *New York City*

IN A paper read before the Second District Dental Society of New York, about thirteen years ago, the use of sodium sulphid solution as a solvent of the epithelial lining of the periodontal pocket was advocated by McCall.[1] Sodium sulphid was said "to have a characteristic solvent action upon epithelial structures," and by removing the epithelial lining of pockets, to facilitate reattachment of the underlying connective tissue to the cementum.

In 1927, the use of sodium sulphid solution was again suggested by Sorrin and Miller.[2,3] They stated that sodium sulphid dissolves epithelial structures in direct proportion to their keratin content; that it attacks hair and nails more readily than epithelial cells and hornified epithelium more readily than non-hornified. Sorrin and Miller stated that sodium sulphid dissolves these tissues chemically and thus facilitates their removal. Rosebury[4] on the other hand, reported that a saturated solution of sodium sulphid produced only swelling, evidently due to imbibition of water, with no evidence of disintegration, on several body surface keratins. According to Karshan[5], keratins are markedly affected by alkaline solutions, "causing swelling and a loss of sulphur, and complete decomposition upon boiling." The boiling, evidently, is most essential for complete disintegration. The recently revised textbook of Stillman and McCall[6], again describes and recommends sodium sulphid solution for removing periodontal pocket epithelium. The technique for the use of the drug, as described in this text, was adhered to in the present investigation.

The formula of the solution used by its advocates and in this work is:

Rx.	Sodium sulphid	70 grains
	Sodium carbonate	20 grains
	Water	1 ounce.

In accordance with the recommendations, before use of this solution in clinical cases, the root surfaces of the teeth were curetted with great care, using instruments such as McCall's planes. This was done to avoid removing or damaging the pocket epithelium before use of the sodium sulphid solution. A slender twist of absorbent cotton, saturated with the solution, was carried to the base of the pocket with a root canal plugger, with care to avoid pressure. Dryness was maintained around the treated teeth by means of cotton rolls, although Stillman and McCall state that dryness is not essential. Stillman and McCall suggest that the twist of cotton and the drug be permitted to remain in the pocket for ten minutes; the cotton is then removed, and "the pocket is preferably left undisturbed for a few minutes to permit more prolonged action of the drug which has penetrated into the soft tissue. A curette is then used in the pocket with the idea of scraping and removing the disintegrated epithelium"[6]. It is suggested that, after curettement, while the blood is clotting, slight pressure be maintained against the external surface of the gingiva.

PROCEDURE:

A. Clinical cases—In order to eliminate uncontrolled effects due to variation in functional stimulation or occlusal stress, the twenty-one patients selected had only their lower six anterior teeth, without opponents. The teeth of each patient were treated on one side (the control) by the conservative method, including the use of curettes to remove the pocket epithelium, and on the other side (the experimental) similarly, except that sodium sulphid was applied before curettes were used to remove the epithelium. The effects of treatment were then observed over a period of sixty-three days. The results were gauged by the shortening and closure of the pockets, along with the disappearance of inflammation and suppuration. In order to determine whether

*Read before the American Academy of Periodontology at its 24th Annual Meeting, Atlantic City, New Jersey, 1937.

†Research from the Department of Periodontology, School of Dental and Oral Surgery, Columbia University.

the pockets actually became more shallow (aside from the effect of gingival shrinkage), each pocket was accurately measured from a permanent mark, in the form of a notch made on the enamel. Then with Box's millimeter measuring probe, held against the explorer, the distance from the notch to the base of the pocket was measured. The pockets were remeasured ten days after treatment and again at about two week intervals thereafter. The final remeasurement of some pockets was made nine weeks after treatment.

B. In vitro controls—In ten patients with teeth condemned for extraction because of advanced periodontoclasia, gingival flaps of the deep pockets were excised. Each excised flap was divided into three pieces, in such a way that each piece bore an analogous area of pocket and surface epithelium. One piece was placed in the sodium sulphid solution, another in sodium hydroxide solution, and the third in physiological saline solution. Each piece of tissue was retained in the respective solution for fifteen minutes, and then immediately placed in Bouin solution. This length of time was chosen since it is the equivalent of that in which the sodium sulphid solution would remain in the pockets, as described earlier. The sodium hydroxide solution was used as a control for the alkalinity of the sodium sulphid solution. The latter, prepared according to the formula given, was found to have a pH of 11.4 by means of a glass electrode potentiometer. A solution was then prepared containing the same percentage of sodium carbonate and enough sodium hydroxide, as determined by titration, to yield the same final pH, 11.4.

C. In vivo controls—In a group of eight patients with teeth condemned for extraction because of advanced periodontoclasia, sodium sulphid solution was inserted into some pockets and sodium hydroxide into others. The solutions of sodium sulphid and sodium hydroxide used were those described above. Curettement of the pockets was not resorted to in these cases. Biopsies were made immediately after the drugs had been permitted to act as described above, after which the teeth were extracted. The pieces of tissue were immediately dropped into Bouin fixing solution and subsequently sectioned for histologic examination.

RESULTS.

A. Clinical cases—The application of the sodium sulphid caused some discomfort to all of the patients, and the marginal gingiva became angry red in color. The alveolar

TABLE I

EFFECT OF CONSERVATIVE TREATMENT ON THE DEPTH OF PERIODONTAL POCKETS, WITH [illegible]COURS WITHOUT APPLICATION OF SODIUM SULPHID SOLUTION

Time	Controls						Treated with Sodium Sulphid					
Post-Operative in Days	No. of Pockets	Average Change of Pockets In MM.		Percentage of Pockets Showing			No. of Pockets	Average Change of Pockets In MM.		Percentage of Pockets Showing		
		Decrease	Increase	Decrease	Increase	No Change		Decrease	Increase	Decrease	Increase	
12	71	0.86		80.2	4.2	15.5	65	0.58		58.4	18.4	
21	64	0.14		42.2	35.9	21.8	57		0.04	33.3	24.5	
23	36		0.06	30.5	22.2	47.2	32		0.09	21.8	43.7	
35	32	0.15		43.7	46.8	9.3	26		0.09	30.7	30.7	
49	12	0.25		50	25	25	15	0.20		60	6.6	
63	8		0.18	12.5	37.5	50	9	0.11		22.2		
Totals	71	0.93		81.7	5.6	12.6	65	0.56		63	18.5	

gingiva and areolar mucosa were not harmed, since they were protected by the cotton rolls. Seventy-one pockets were used as controls, in which the drug was not used. Remeasurement of these pockets showed an average decrease in depth of nearly one millimeter per pocket. Of these pockets, 81.7% showed decrease in pocket depth; 5.6% showed increase in pocket depth and 12.6% showed no change. Sixty-five pockets were treated with sodium sulphid. These showed an average decrease in depth of ½ millimeter per pocket. 63% of these pockets showed decrease in pocket depth, as against the 81.7% of the pockets on the control side; 18.5% showed increase in pocket depth and 18.5% no change. (Table I.)

Table II represents a typical case which shows that four pockets on the control totalled *a decrease of pocket depth* of five millimeters, whereas the total change on the sodium sulphid side, was an *increase in pocket depth* of ¼ millimeter. Table III shows another typical case in which the total changes in both control and sodium sulphid pockets are approximately the same, although remeasurement at the end of ten days indicated an improvement in all six pockets on the control side, whereas only three of the five pockets on the sodium sulphid side were decreased in pocket depth.

B. In vitro controls—The histologic sections of the gingival tissue which had been immersed in sodium sulphid solution showed disintegration of the corneal layer of the epithelium. (It may be noted that no corneum was observed on the pocket epithelium in the sections.) A similar effect was noticed on a small portion of the prickle cell layer of both the outer and pocket epithelia. In this portion the prickle cells were somewhat swollen and were poorly stained. In some instances cytoplasm and nuclei were completely disorganized; only the cell membranes remained. However, the bulk of the prickle cell layer was intact, and the basal cells of the epithelium were firmly attached to the underlying connective tissue.

It is interesting to note that the sodium sulphid solution also disintegrated that portion of the connective tissue surfaces, which came in direct contact with the drug, as evidenced by poor staining of the superficial tissue as contrasted with the underlying

TABLE II.

COURSE OF TREATMENT OF PERIODONTAL POC KETS, WITH AND WITHOUT SODIUM SULPHID, IN A TYPICAL CASE. (CASE 2) *

Time	*Controls*					*Treated with Sodium Sulphid*						
Post-Operative in Days	Pockets A	B	C	D	Totals	Pockets E	F	G	H	J	K	Totals
10	−2	−2	−1	0	−5	−1	0	0	+1	0	−1	−1
17	−¾	0	−¼	−¾	−1¾	+½	0	0	−¼	−½	−½	−¾
24	+½	0	+½	+½	+1½	+¼	+¼	−¼	+¼	+½	+¼	+1¼
32	+¾	0	−¼	−¾	−¼	+¾	0	−¼	0	+1	0	+½
67	−½	+¾	0	+¼	+½	−1	0	+¼	−¼	−¼	0	−1¼
Totals	−2	−1¼	−1	−¾	−5	−½	+¼	−¼	+¾	+¾	−1¼	+¼

The minus and plus signs indicate decrease and increase, respectively, in depth of pocket in millimeters. For example −2 refers to a decrease in pocket depth by 2 millimeters.

normal tissue, and by disorganization of both cells and fibres. All the changes noted in the epithelial and connective tissues with the use of sodium sulphid solution, were also found in the sections treated with sodium hydroxide, with one exception, in that the corneum was disintegrated less by the sodium hydroxide (Figures 1-10).

C. In vivo controls—The gingival tissue sections studied *in vivo* with sodium sulphid showed similar changes in the epithelium as in the *in vitro* controls, with the exception that the connective tissue was not affected, because the drug disintegrated only the surface layer of the prickle cells and did not penetrate to the connective tissue. Here again similar changes were produced by the sodium hydroxide. In the absence of corneum on the pocket epithelium, the changes produced by the sodium sulphid and the sodium hydroxide solutions were indistinguishable.

DISCUSSION.

The results obtained with sodium sulphid solution in the clinical cases do not support the view of McCall,[1] Sorrin and Miller,[2,3] and Stillman and McCall[4] as to the value of this drug in the treatment of periodontal pockets. The findings indicate, on the other hand, that sodium sulphid may be detrimental in treatment, since the average de-

Fig. 1—Section of gum immersed in physiological saline solution *in vitro*. Outer epithelium is shown: (a) corneum; (b) prickle cell layer; (c) connective tissue. All layers of the epithelium were unaffected by the saline solution.

TABLE III.

COURSE OF TREATMENT OF PERIODONTAL POCKETS, WITH AND WITHOUT SODIUM SULPHID IN A TYPICAL CASE. (CASE 8) *

Time	*Controls*							*Treated with Sodium Sulphid*					
Post-Operative in Days	Pockets A	B	C	D	E	F	Totals	Pockets G	H	J	K	L	To
10	−1	−2	−1¾	−1	−1¼	−¾	−7¾	−1¼	−¼	−2½	0	0	-4
14	+¾	−¼	−½	0	+½	+¼	+¾	+¾	0	−1	−¾	0	-1
19	−¾	0	0	0	0	−¼	−1	−¾	−¾	+¾	−½	0	-1
26	+¼	−¼	−¼	−¼	+¼	+¼	0	+¾	+¾	0	+¼	0	+1
40	−1¼	+¼	0	0	−½	−¼	−1¾	−¼	−½	−¼	−¼	0	-1
64	+½	0	+½	0	+¾	0	+1¾	−¾	0	0	0	0	-¾
Totals	−1½	−2¼	−2	−1¼	−¼	−¾	−8	−1½	−¾	−3	−1¼	0	-6

* The minus and plus signs indicate decrease and increase, respectively, in depth of pocket millimeters. For example −2 refers to a decrease in pocket depth by 2 millimeters.

crease in pocket depth was nearly twice as great with simple curettement (in the control pockets) as it was with curettement supplemented by application of sodium sulphid solution.

The histological studies of the effects of sodium sulphid solution on the gingival tissues, both in the mouth and *in vitro*, show that the destructive effects of the solution are due almost entirely to its strong alkalinity and are largely independent of the sulfhydryl and sulphid ions. This follows from the finding that all but one of the effects observed with sodium sulphid were obtained in equal degree with a solution containing sodium hydroxide in place of sodium sulphid and having the same pH, 11.4, as the sodium sulphid solution. The exception consisted in the fact that the corneal layer of the outer epithelium was more markedly affected by the sodium sulphid solution. The

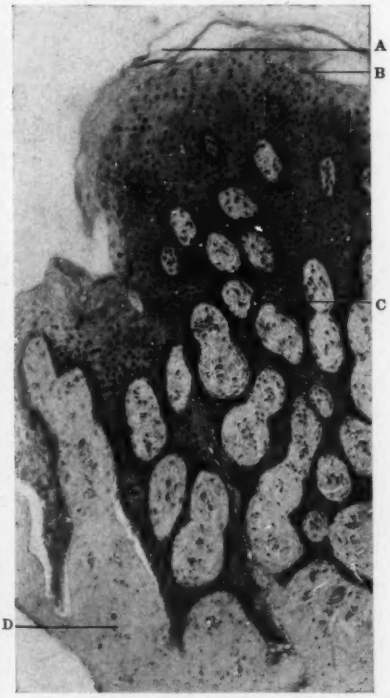


Fig. 2—Section of gum immersed in sodium sulphid *in vitro*. Outer epithelium is shown: (a) disintegrated corneal layer; (b) prickle cells directly beneath corneum are swollen and take stain poorly; (c) remainder of epithelium is unaffected; (d) connective tissue at exposed surface takes stain poorly.

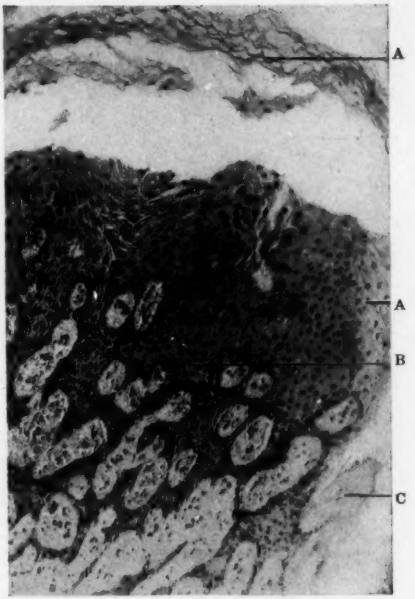


Fig. 3—Section of gum immersed in sodium sulphid *in vitro*. Pocket epithelium is shown: (a) surface prickle cell layer is disintegrated; the cytoplasm and nuclei of the cells are completely disorganized; (b) remainder of epithelium is unaffected; (c) exposed surface of connective tissue takes stain poorly.

Fig. 4—Section of gum immersed in sodium sulphid *in vitro*. Pocket epithelium is shown: (a) surface prickle cell layer is disintegrated; the cytoplasm and nuclei of the cells are completely disorganized; (b) remainder of epithelium is unaffected; (c) exposed surface of connective tissue takes stain poorly.

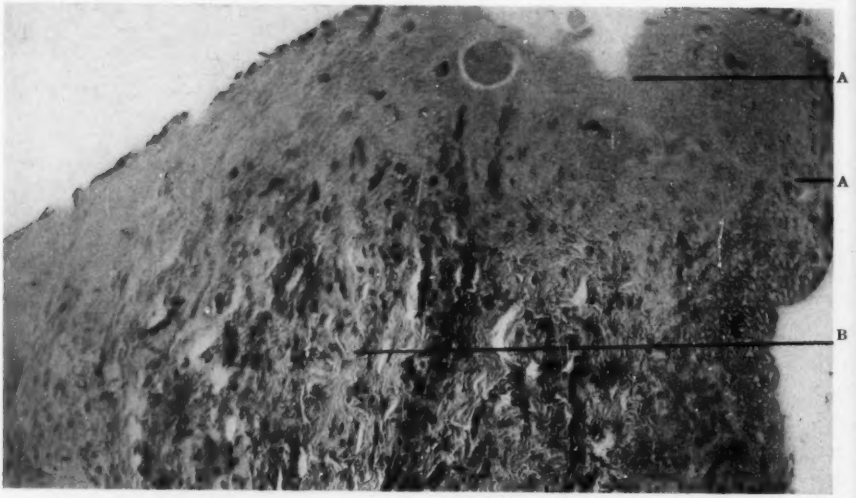


Fig. 5—Section of gum immersed in sodium sulphid *in vitro*. Connective tissue is shown: (a) tissue takes stain poorly; the cellular and extracellular material has been disintegrated; (b) tissue distant from the sodium sulphid unaffected.

sodium hydroxide solution was prepared so as to be identical with the sodium sulphid solution except that the latter contained sulfhydryl ions and sulphid ions. It is therefore evident that only the extra effect on the corneum of the outer epithelium can be attributed to the sulfhydryl and sulphid ions.

Both solutions tested disintegrated only superficial cell layers of the pocket epithelia.

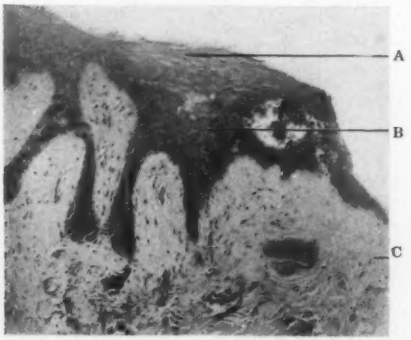


Fig. 6—Section of gum immersed in sodium hydroxide *in vitro*. Pocket epithelium is shown: (a) disintegrated surface of prickle cell layer; the cells are swollen, take stain poorly and nuclei and cytoplasm are disorganized; (b) remainder of epithelium unaffected; (c) exposed surface of connective tissue takes stain poorly.

This epithelium lacks a cornified layer, and the sodium sulphid solution had no more pronounced effect upon it than did the sulphid-free solution of equal alkalinity

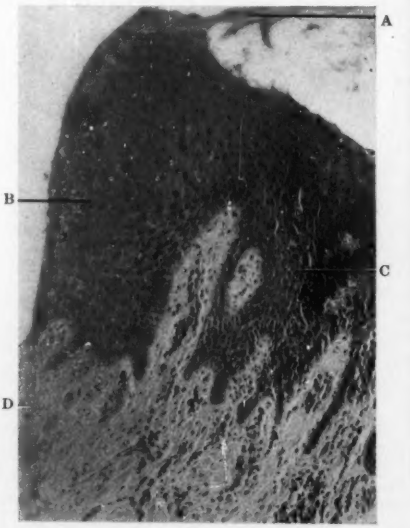


Fig. 7—Section of gum immersed in sodium hydroxide *in vitro*. Outer epithelium is shown: (a) corneum partly disintegrated; (b) prickle cells are swollen and take stain poorly; (c) remainder of epithelium is unaffected; (d) exposed surface of connective tissue takes stain poorly.

(pH). The effect observed in other words was due to the caustic action of alkali and not to the sulfhydryl and sulphid ions.

SUMMARY.

1. Sodium sulphid solution, prepared and used clinically according to the directions of Stillman and McCall, was found to confer no advantage over careful curettement in the treatment of periodontal pockets. Pockets treated by curettement alone showed an average decrease in depth during the period of treatment nearly twice as great as those treated by curettement supplemented by the application of sodium sulphid solution.

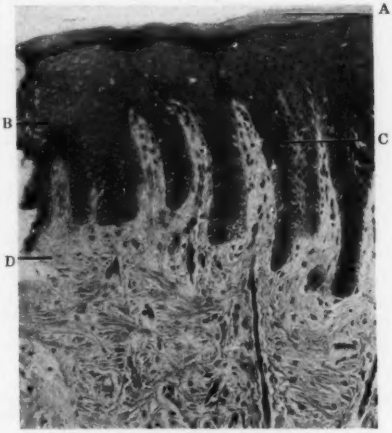


Fig. 8—Section of gum immersed in sodium hydroxide *in vitro*. Outer epithelium is shown: (a) corneum is only slightly affected; (b) prickle cells swollen and take stain poorly; (c) remainder of epithelium unaffected; (d) exposed surface of connective tissue partly affected in its ability to take stain.

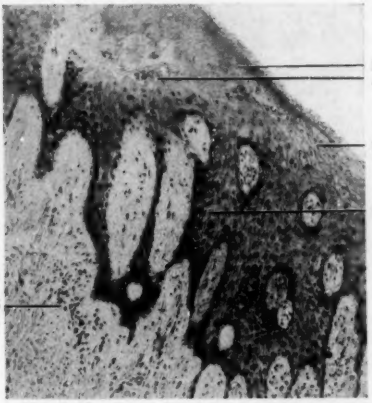


Fig. 9—Section of gum treated with sodium sulphid *in vivo*. Pocket epithelium is shown: (a) surface prickle cell layer is disintegrated; the cytoplasm and nuclei of the cells are completely disorganized; (b) prickle cells are swollen; (c) remainder of epithelium is unaffected; (d) blood; (e) unaffected connective tissue.

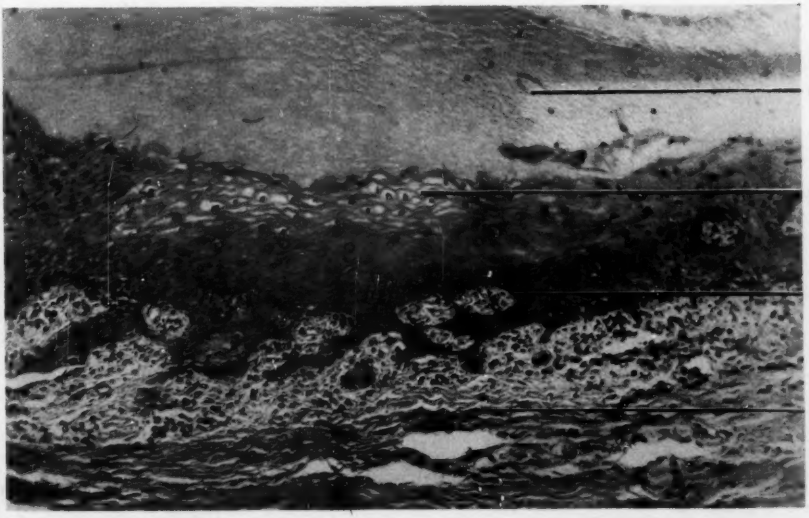


Fig 10—Section of gum treated with sodium hydroxide *in vivo*. Pocket epithelium is shown: (a) surface prickle cell layer undergoing disintegration; (b) remainder of epithelium is unaffected; (c) blood; (d) unaffected connective tissue.

2. The alkaline sulphid solution as employed destroys only the superficial layers of the epithelium, leaving the prickle and basal cells largely intact. Hence the principle upon which the use of the sodium sulphid solution is based—to obtain complete disintegration of the pocket epithelium in order to facilitate its removal and obtain reattachment of the connective tissue to the cementum—is unfounded.

3. The destructive effects of the sodium sulphid solution, applied in the mouth or on excised pieces of periodontally affected gingiva and studied in histological sections, were duplicated almost entirely by similar application of a solution of equal alkalinity (pH) but lacking sodium sulphid. The sulphid solution had a more marked effect only on the corneal layer of the outer epithelium. This effect is of no value since there is no corneal layer in pocket epithelia.[4] The effect of sodium sulphid solution on the pocket epithelia was evidently due entirely to its strong alkalinity and independent of its content of sulfhydryl and sulphid ions. Since the sulfhydryl and sulphid ions themselves do not appreciably affect the non-cornified pocket epithelium, its use in the treatment of periodontal pockets appears to be irrational as well as ineffective.

The writer wishes to acknowledge his gratefulness to Doctors Theodor Rosebury and Isador Hirschfeld, for their many suggestions and for the interest and guidance which they maintained throughout this study; and to Doctor Maxwell Karshan for his aid.

REFERENCES

1. McCall, J. O.: *D. Items Interest*, **48**: 342, 1926.
2. Sorrin, S. and Miller, S.: *D. Cosmos*, **69**: 1113, 1927.
3. Sorrin, S. and Miller, S.: *The Practice of Periodontia*; the MacMillan Co., New York, 1928, p. 96.
4. Rosebury, T.: *J. D. Res.*; **10**: 187, 1930.
5. Karshan, M.: *J. D. Res.*; **10**: 181, 1930.
6. Stillman, P. R., and McCall, J. O.: *A Textbook of Clinical Periodontia*; the MacMillan Co., New York, 1937, p. 219.

630 West 168th Street

ABBREVIATIONS*

for

Dental Periodicals Published in the United States and Canada

Approved by the

AMERICAN ASSOCIATION OF DENTAL EDITORS

	TITLE ABBREVIATION
The Alpha Omegan	*Alpha Omegan*
The Angle Orthodontist	*Angle Orthodont.*
Annals of Dentistry	*Ann. Den.*
The Apollonian	*Apollonian*
Archives of Clinical Oral Pathology	*Arch. Clin. Oral Path.*
The Articulator of the St. Louis University Dental Alumni Association	*Articulator St. Louis Univ. D. Alumni A.*
Bulletin of the American Society of Oral Surgeons and Exodontists	*Bul. Am. Soc. Oral Surg. & Exodont.*
The Bulletin of the Chicago Dental Society	*Bul. Chicago D. Soc.*
Bulletin of the Cincinnati Dental Society	*Bul. Cincin. D. Soc.*
Bulletin of the Milwaukee County Dental Society	*Bul. Milwaukee Co. D. Soc.*
Bulletin of the Nassau County Dental Society	*Bul. Nassau Co. D. Soc.*
Bulletin of the Ninth District Dental Society	*Bul. 9th Dist. D. Soc. (N. Y.)*
Bulletin of the North Carolina State Dental Society	*Bul. N. Car. D. Soc.*
Bulletin of the Odontological Society of Western Pennsylvania	*Bul. Odont. Soc. West. Pa.*
The Bulletin of the Oklahoma State Dental Society	*Bul. Okla. D. Soc.*
The Bulletin of the Pacific Coast Society of Orthodontists	*Bul. Pacific Coast Soc. Orthodont.*
The Bulletin of the Philadelphia County Dental Society	*Bul. Phila. Co. D. Soc.*
The Bulletin of the Virginia State Dental Association	*Bul. Va. D. A.*
Bushwacker	*Bushwacker*
Caementum	*Caementum*

*Extract from Report of Committe on Abbreviations A. A. D. E. *Transactions* Eighth Annual Meeting, 1938—pp. 53-54.

	TITLE ABBREVIATION
Central Dental News	*Central D. News*
The Columbia Dental Review	*Columbia D. Rev.*
Connecticut State Dental Association Bulletin	*Conn. D. A. Bul.*
Contact Point	*Contact Point*
The Datter	*Datter*
Delta Sigma Delta Desmos	*Desmos*
The Dental Assistant	*D. Asst.*
Dental Craftsman	*D. Craftsman*
Dental Digest	*D. Digest*
Dental Hygiene Quarterly	*D. Hyg. Quar.*
Dental Items of Interest	*D. Items Interest*
Dental Laboratory Review	*D. Lab. Rev.*
Dental Outlook	*D. Outlook*
Dental Radiography and Photography	*D. Radiog. & Photog.*
Dental Rays	*D. Rays*
Dental Students' Magazine	*D. Students' Mag.*
Dental Survey	*D. Survey*
Dentogram	*Dentogram*
Dentoscope	*Dentoscope*
The Detroit Dental Bulletin	*Detroit D. Bul.*
Essex County Dental Society Bulletin	*Essex Co. D. Soc.*
The Frater of Psi Omega	*Frater*
Georgetown Dental Journal	*Georgetown D. J.*
Harvard Dental Record	*Harvard D. Rec.*
Hudson County Dental Society Bulletin	*Hudson Co. D. Soc. Bul. (N. J.)*
The Illinois Dental Journal	*Ill. D. J.*
Impressions	*Impressions*
International Journal of Orthodontia and Dentistry for Children	*Internat. J. Orthodont. & Den. Children*
Iowa Dental Bulletin	*Iowa D. Bul.*
The Journal of the American College of Dentists	*J. Am. Col. Den.*
The Journal of the American Dental Association	*J. A. D. A.*
The Journal of the American Dental Hygienists Association	*J. Am. D. Hygienists A.*
The Journal of the Arkansas State Dental Association	*J. Ark. D. A.*
The Journal of the Baltimore College of Dental Surgery	*J. Balt. Col. D. Surg.*
The Journal of the California State Dental Association	*J. Calif. D. A.*
The Journal of the Canadian Dental Association	*J. Canad. D. A.*
Journal of the Colorado State Dental Association	*J. Colo. D. A.*
The Journal of Dental Education	*J. D. Educ.*
Journal of Dental Research	*J. D. Res.*
Journal of the District of Columbia Dental Society	*J. Dist. of Columbia D. Soc.*
The Journal of the Florida State Dental Society	*J. Florida D. Soc.*
The Journal of the Houston District Dental Society	*J. Houston Dist. D. Soc.*
The Journal of the Indiana State Dental Association	*J. Ind. D. A.*
The Journal of the Kansas State Dental Association	*J. Kan. D. A.*
The Journal of the Michigan State Dental Society	*J. Mich. D. Soc.*
The Journal of the Missouri State Dental Association	*J. Mo. D. A.*
The Journal of the Nebraska State Dental Association	*J. Neb. D. A.*
The Journal of the New Jersey State Dental Society	*J. N. J. D. Soc.*
Journal of the Ohio State Dental Society	*J. Ohio D. Soc.*
The Journal of the Oregon State Dental Association	*J. Ore. D. A.*
The Journal of Periodontology	*J. Periodont.*
The Journal of the Second District Dental Society	*J. 2nd Dist. D. Soc.*
The Journal of the Southern California State Dental Association	*J. South. Calif. D. A.*
The Journal of the Wisconsin State Dental Society	*J. Wis. D. Soc.*
Kings County Dental Society Bulletin	*Kings Co. D. Soc. Bul. (N. Y.)*
McGill Dental Review	*McGill D. Rev.*
The M. D. S. Bulletin	*M. D. S. Bul. (N. Y.)*
Medentian	*Medentian*
The Minneapolis District Dental Journal	*Minneap. Dist. D. J.*
Modern Dentistry	*Mod. Den.*
Mouth Health Quarterly	*Mouth Health Quar.*
The Newark Dental Club Bulletin	*Newark D. Club Bul.*
The New York Journal of Dentistry	*N. Y. J. Den.*
Northwest Dentistry	*Northwest Den.*
Northwestern University Bulletin	*Northwest. Univ. Bul.*
Nutrition and Dental Health	*Nutrition & D. Health*
The N. Y. U. Dental News	*N. Y. U. D. News*
Oral Hygiene	*Oral Hyg.*
The Penn Dental Journal	*Penn D. J.*
The Pennsylvania State Dental Journal	*Pa. D. J.*
Proceedings of the American Association of Dental Schools	*Proc. Am. A. D. Schools*
Review of Dentistry for Children	*Rev. Den. Children*
Squibbs Memoranda	*Squibbs Memo.*
St. Louis Dental Society Bulletin	*St. Louis D. Soc. Bul.*
Standard Dental Topics	*Standard D. Topics*
Temple Dental Review	*Temple D. Rev.*
The Texas Dental Journal	*Texas D. J.*
The Tufts Dental Club of New York	*Tufts D. Club Bul. (N. Y.)*
Tufts Dental Outlook	*Tufts D. Outlook*

University of Illinois College of Dentistry Alumni Bulletin	*Univ. Ill. Col. Den. Alumni Bul.*
Washington State Dental Journal	*Wash. D. J.*
The Washington University Dental Journal	*Wash. Univ. D. J.*
West Virginia Dental Journal	*W. Va. D. J.*
Xi Psi Phi Quarterly	*Xi Psi Phi Quar.*
Year Book Alumni Association, College of Dentistry, U. S. C.	*Year Book Alumni A., Col. Den., Univ. South. Calif.*

GENERAL COMMENT ON ABBREVIATIONS

1. States of the Union be abbreviated as conventional usage decrees.
2. Cities be abbreviated when usage permits, as in Phila., Bost., San Fran., Cincin., Wash., Minneap., and Balt.
3. State districts be reduced to the numeral, as *J. 2nd Dist. D. Soc.*
4. Titles of foreign periodicals be written in full. However, if necessary to abbreviate, then follow the abbreviations used in the *Quarterly Cumulative Index Medicus* of the A. M. A. A list will be found in any issue.
5. For abbreviations of titles of medical and scientific periodicals, where the basic ones are not included in the present list, consult the *Quarterly Cumulative Index Medicus* listing.
6. Where confusion might result, write states and other geographical names in full. It is suggested also that where a title abbreviation, even in combination, appears obscure or confusing, write in full.
7. As an aid for the attainment of uniformity, the following is suggested: that all dental periodicals print in some conspicuous place on their pages, the accepted abbreviation for their particular periodical. This might appear on the cover, the contents page, the mast-head, with the index, with the running volume data, or some such similar and easily seen location.
8. In articles intended for lay reading, do not abbreviate.
9. It should be kept in mind that nearly all abbreviations in combinations, are clear, even when very brief, and that such a combination will almost automatically suggest the full title.

LIST OF BASIC ABBREVIATIONS *(American dental periodicals)*

Abstract ---------- Abstr.	Eastern ---------- East.	Proceedings ---------- Proc.
Academy ---------- Acad.	Education ---------- Educ.	Public ---------- Pub.
Alumni ------ not abbreviated	Experimental ---------- Exper.	Quarterly ---------- Quar.
America ---------- Am.*	Gazette ---------- Gaz.	Radiography ---------- Radiog.
Annals ---------- Ann.	Graduate ---------- Grad.	Radiology ---------- Radiol.
Archives ---------- Arch.	Health ------ not abbreviated	Record ---------- Rec.
Assistant (s) ---------- Asst.	Hygiene ---------- Hyg.	Report ---------- Rep.
Association ---------- A.	Institute ---------- Inst.	Research ---------- Res.
Bulletin ---------- Bul.	International ---------- Internat.	Review ---------- Rev.
Bureau ---------- Bur.	Journal ---------- J.	Science ---------- Sc.***
Canadian ---------- Canad.	Laboratory ---------- Lab.	Scientific ---------- Scient.
Clinical ---------- Clin.	Magazine ---------- Mag.	Section ---------- Sect.
College ---------- Col.	Medical ---------- M.	Society ---------- Soc.
Congress ---------- Cong.	Medicine ---------- Med.	South ---------- S.
Cosmos ------ not abbreviated	Modern ---------- Mod.	Southern ---------- South.
County ---------- Co.	National ---------- Nat.	Supplement ---------- Suppl.
Dental ---------- D.	North ---------- N.	Surgery ---------- Surg.
Dentistry (Denista) ---- Den.	Northern ---------- North.	Transactions ---------- Tr.
Department ---------- Dept.	Odontological ---------- Odont.	University ---------- Univ.
Digest ------ not abbreviated	Orthodontia (ics-ist) Orthodont.**	West ---------- W.
District ---------- Dist.	Photography ---------- Phctog.	Western ---------- West.
East ---------- E.		

*Except when it refers to official publications of the American Dental Association, then the simple A. only. As, *J. A. D. A.*

**If in a long combination where meaning will be clear, use Orth.

***When reference is being made to the magazine *Science* do not abbreviate—write in full.

REFERENCES AND CITATIONS

Periodical citations:

The method proposed is consistent with that employed by the *Quarterly Cumulative Index Medicus*, and that suggested by Hackh. The reference gives, in the following order:

1. The name of the author
2. The title of the article
3. The abbreviation of the title of the periodical *(ital.)*
4. The volume (bold-face type, Arabic numeral)
5. The paging
6. The month (day of month if a semi-monthly, semi-weekly, or weekly publication)
7. The year

Book citations:

In this instance, reference has been made to the usages of the University of Chicago Press:

1. The name of the author
2. The title of the book *(italic)*
3. The edition, if more than one
4. Place of publication
5. Publisher
6. Date
7. Paging

(For further details concerning the usages in conjunction with periodical and book citations, refer to the 1937 Report as published, and mentioned previously in this present report.)

BOOK REVIEWS

Dental Histology and Embryology Including Laboratory Directions. By Frederick Bogue Noyes, B. A., D. D. S., Sc. D., F. A. C. D., Professor of Histology, Northwestern University Dental School, 1896-1914; Professor of Histology and Orthodontia, College of Dentistry, University of Illinois, 1914. Fifth Edition, Revised and largely rewritten. By Isaac Schour, B. S., D. D. S., M. S., Ph. D. Professor of Histology and Head of Department, University of Illinois, College of Dentistry. And Harold Judd Noyes, B. S., D. D. S., M. D., Clinical Assistant Pediatric Department, Rush Medical College, 1935; Associate 1936; Assistant Pediatrician on Staff at Presbyterian Hospital, Clinic, University of Chicago, 1937. Philadelphia, Lea & Febiger 445 pages, 284 illustrations. 1938.

This fine book, though nominally the fifth edition of Dr. Frederick B. Noyes' *Dental Histology and Embryology,* first published in 1914, is virtually a new work by his son Dr. Harold Judd Noyes and Dr. Isaac Schour. It is practically impossible to give an adequate idea of a book of such scope and importance within the limits of a brief review; each subject is treated with much careful and exact detail and must be read to be fully appreciated.

The early chapters have to do with embryology, the first being a consideration of general principles of cell differentiation and development, following this the prenatal development of the face and oral cavity, then that of the tooth germ itself.

In succeeding chapters the various structures of the oral cavity are taken individually. Their histogenesis, histology, development, physical and chemical characteristics, distribution, and biologic relations are described with meticulous scientific detail, but the substance of this part of the text conforms in the main to the works of other prominent authorities.

There are exceptionally full chapters on enamel and dentin, in fact, if any criticism were to be made, it would be that the authors have over-emphasized these structures in comparison with the discussion of the supporting tissues. "Histogenetically, histologically, chemically, and physically, enamel stands alone"; from its earliest beginnings in the cells of the ectoderm it differs from other calcified tissues, all others deriving from the mesoderm. The ameloblasts being lost and destroyed when enamel is completed, the authors are unequivocal in their belief that finished enamel has no vitality and "any such thing as vital change in the tissue is unthinkable." It is not subject to calcium withdrawal; once formed and calcified it can be altered only by chemical and physical changes in the environment and has no power to repair or regenerate damaged cells. Enamel should not be compared with bone which, because of its histological and biological relations, can be resorbed and rebuilt and which serves as a storehouse for calcium subject to withdrawal.

To a great degree the same facts hold good for dentin. After its extra-cellular matrix has been formed and calcified, it too can be changed only through actual destruction from external causes such as caries, surgical removal, etc., and unlike bone it is not subject to calcium withdrawal.

The authors again and again emphasize the distinction between actively growing enamel and dentin and that which is adult and fully formed. During their calcifying period these structures are extremely sensitive to fluctuations in the metabolic processes, particularly calcium metabolism.

Caries of the teeth is an active destruction by outside agencies, of a formed material which is the result of cell activity, the teeth themselves being passive. The cellular activities of organs and tissues of the body have an influence, but this is only in producing those conditions of environment which render the activities of the destructive agent efficient in their action upon the tooth tissues.

The authors have endeavored to vitalize the text by connecting structure and function, correlating the knowledge of microscopic anatomy with physiologic and clinical findings and the whole with practical operative procedures. Especially in the treatment of the enamel walls of cavities it is constantly necessary to have and to apply a knowledge of minute anatomy. It is probably not too much to say that nowhere

else in the literature can a more careful exposition of this matter be found.

The authors make an additional classification of cavities on the basis of enamel structure: (1) those in which the enamel rods are inclined towards the cavity; (2) those in which the rods are inclined away from the cavity.

The importance of cementum function is stressed. It is built continuously by the cells of the periodontal membrane. Teeth move microscopically throughout life and new layers of cementum for attachment of new periodontal fibres are necessary to conform to changed strains. Tooth retention depends on this.

The authors argue that teeth from which pulps have been removed cannot be called dead when they remain in normal function and continue to appose new layers of cementum.

The subject matter on periosteum is taken largely from G. V. Black's work (1887).

The "Histo-physiological Remarks" throughout the text will be found particularly interesting. Under this head in the chapter on alveolar process there is a discussion of the mesial drift of the teeth with accompanying resorption and apposition of alveolar bone in response to the pressure and tension respectively of the periodontal fibres.

> This constant adjustment reveals the dynamic quality of bone. The alveolar process is not a static support of the teeth, and its structure and position is determined by the functional demand of the constantly moving dentition. The delicate balance, between the formation and destruction of the alveolar process, is swayed in one direction or the other by environmental pressure stimuli. Only when the stresses are beyond the tolerance of the supporting tissues, which of course varies with age and fluctuating degrees of health and disease, do we find destruction when support is demanded.

The chapters on eruption of the teeth and postnatal development of the face and oral cavity contain much that is significant and clinically important. These subjects have been brought quite up-to-date by the inclusion of the work of Logan and Kronfeld and Schour on eruption and that of Schour on growth patterns of the teeth.

> Histological examination reveals a pattern and gradient of growth in the form of normal incremental rings which is characteristic for each tooth type . . . Histological analysis of teeth following various constitutional disturbances shows that each particular dysfunction produces a characteristic record which is superposed on the basic pattern in the form of accentuated incremental rings of arrested growth or disturbed calcification. The neonatal ring, for example, reflects the physiological readjustments incident to birth,

and metabolic changes at this time are permanently recorded in the enamel and dentin of deciduous teeth. This particular kymographic record is termed the neonatal ring.

The enamel is divided by the neonatal ring into two sharply defined zones, the prenatal more homogeniously and densely calcified than the postnatal probably because of the greater protection and nourishment given the foetus in utero. Schour's work with alizarine in measuring tooth growth is a development of Hunter's use of madder in the study of bone apposition.

A most interesting modification of McBeath's diagram of the chronology of the deciduous teeth, shows in addition the position of the neonatal line. Another interesting diagram in this connection is Schour's frequently "quoted" one of the incremental lines of calcium apposition in the tooth.

The outstanding work of Broadbent and the late Wingate Todd (of the Bolton Foundation) has been included in the chapter on postnatal development along with some of their cephalometric roentgenograms and tracings. A fine chart summarizes postnatal dental development. The period of mixed dentition is hazardous because of the change and intensity of growth forces and the loss of reciprocal tooth support through the shedding of deciduous teeth; also there is a relative frequency of systemic disease at this time. The reflex nervous disturbances of the eruptive period are explained.

The chapter on comparative Dental Histology is new, and interesting in its implications as well as the factual content. The authors have graphically described the evolution of the teeth as they conform in shape and structure to gradually increasing functions, starting as organs of prehension, becoming later instruments of combat, then tools and finally masticatory organs. The development of modes of tooth implantation from the simple placoid scale having no relation to the adjacent bone, to the ultimate arrangement of the human tooth which we know as gomphosis, is also set forth. Function of teeth determines form and position, and wherever diet and function are similar in animals tooth forms are also similar even in widely separated localities. Teeth of all animals are composed of enamel, dentin, and pulp and all have a dual deriva-

tion from epithelial and connective tissue sources. There are, however, microscopic differences and variations in hardness dependent on the work they perform.

Part Two, entitled Directions for Laboratory Work, consists of some 30 pages devoted to laboratory exercises for students.

Excellent illustrations throughout the book clarify the text, the authors' own as well as many fine ones from the works of Black, Kronfeld, Orban and others.

A comprehensive index, and the extensive bibliography at the close of each chapter add measurably to the value of the book.

Altogether this volume is a very important addition to any dentist's library.

—*Celia Rich*

Biology and Pathology of the Tooth and Its Supporting Mechanism. By Bernhard Gottlieb, Research Professor, Columbia University Dental School, Formerly Professor, University of Vienna Dental School, and Balint Orban, Assistant Professor, Northwestern University Dental School, Chicago, Formerly Professor, University of Vienna Dental School. Translated and edited by Moses Diamond, Associate Professor, Columbia University Dental School, New York, Head of Dental Anatomy Department. Pp. 195 with 166 illustrations. Cloth. New York: The Macmillan Company. 1938.

Bernhard Gottlieb and his assistant, Balint Orban, in 1933 published in German a summary of their investigations on the histology and pathology of the teeth and their investing tissues, including a description of their technique of scaling, brushing of the teeth, cavity preparation, and restorations to replace lost teeth. This book was very popular in Europe, and the second edition was printed in 1936.

Fortunately for the English-speaking men of the profession, M. Diamond, Professor of Dental Anatomy, Columbia University, undertook the work of translating this book. In doing so, he wisely omitted the part on clinical procedure, for many of the technical procedures described in the book are quite different from those used in this country. The English book, *Biology and Pathology of the Tooth and Its Supporting Mechanism,* is an excellent, compact representation of the basic findings and views of Gottlieb and his collaborators. It summarizes the scientific findings contained in more than two hundred and fifty individual articles and publications which have appeared in scientific journals throughout the world, and clearly depicts the physiologic and pathologic changes that occur in the dental and periodontal tissues throughout life.

The book is divided into the following major chapters or subjects: Biology of the Supporting Mechanism of the Tooth, which includes, among other subjects, eruption, gingival crevice, and the biology of cementum, periodontal membrane, and alveolar bone; Accelerated Tooth Eruption; Gingival Inflammation, Schmutz-Pyorrhea, dealing with the etiology and histopathology of gingivitis; Parodontal Pyorrhea, describing the pathology of the deep, suppurating pockets; Diffuse Atrophy of the Alveolar Bone, including pathologic wandering and other symptoms and problems associated with diffuse atrophy; Traumatic Occlusion, containing the histologic changes induced by occlusal overstress, the effects of lateral pressure, the question of tissue tolerance, and other related problems.

At the end of the book is a complete bibliography of the publications of Gottlieb, Kellner, Kotanyi, Kronfeld, Orban, Schwarz, Stein, Weinmann, and others who worked in Gottlieb's laboratory. There is also a glossary containing the German dental words used in the original text.

The book has been well printed; the illustrations especially are noteworthy. Years ago Orban was one of the first to make microphotography an art; later he introduced his excellent technique in this country. The photomicrographs of this book are proof of its excellence.

In spite of the fact that it contains little besides the findings and viewpoint of one group of investigators, this translation is a most valuable contribution to the literature of periodontology.

—*Rudolf Kronfeld*

A Text Book of Histology. By Alexander A. Maximow, Late Professor of Anatomy, University of Chicago, and William Bloom, Associate Professor of Anatomy, University of Chicago, Third Edition; completely revised. Some in colors. Philadelphia and London: W. B. Sanders Company. 1938.

668 pages, 542 illustrations. Review copied from *J. A. D. A.* 26(1). Jan. 1939.

The textbook of histology of Maximow and Bloom needs no introduction. In less than a decade, it has established itself as one of the leading texts for teaching and reference. The fundamentals are clearly stated and beautifully illustrated. Contrary to the old-time type of histology book, which was confined to stereotyped descriptions of the microscopic appearance of cells and tissues, the new Maximow-Bloom book contains many additional discussions of the normal and pathologic physiology of the body tissues. Under the heading "Histophysiologic Remarks," function and clinical correlations are considered and illustrated. The chapter on the teeth is brief, but contains essential and up-to-date information. The chapters on epithelium, connective tissue, and bone should be consulted by any dentist who wishes to familiarize himself with the fundamentals of the most important oral tissues.

—*R. Kronfeld*

Accepted Dental Remedies: Containing a list of official drugs selected to promote a rational dental materia medica and descriptions of acceptable nonofficial articles, by Council on Dental Therapeutics, American Dental Association, 1938.

In scope, form and purpose the new edition of Accepted Dental Remedies is similar to those prepared previously under the direction of Dr. Samuel M. Gordon, the former Secretary of the Council, the first of which was published in 1934.

The primary purpose of the Council has been the gathering and disseminating of information that will protect the dental profession in prescribing and in the use of proprietary substances; and for the purpose of rationalizing dental therapeutics. They list and describe "those pharmaceutical articles which stand acceptable to the Council on Dental Therapeutics. This list includes those official drugs and preparations which are considered to be of greatest usefulness in the field of dentistry, and nonofficial articles marketed in conformity with the rules of the Council."

In the 1938 volume the articles which have been accepted since the last edition and which have been described in the Journal of the A. D. A. since then have of course been added.

Errors have been corrected that have appeared in earlier editions and the text has been brought up-to-date by additions and alterations to make certain parts of the text conform to latest scientific developments. For example, an article on Cevitamic acid has been added and the chapter on Vaso constrictors has been revised.

The Council on Dental Therapeutics of the American Dental Association is doing a fine piece of work and the results are made available to the profession in convenient and amazingly condensed form in these annual volumes.

To quote the Journal Editor's comment, July 1935, "Accepted Dental Remedies is a book which periodontists especially find most useful. The information concerning the ingredients of dentrifrices and mouth washes with which every dentist should be conversant is presented in a readable, time-saving manner. The 'bibliographical index to proprietary and unofficial articles not included in A. D. R.' is worth the small cost of the book and no periodontist can afford to be without this up-to-date little volume."

—*Rosalie Carter*